IN THE UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF TENNESSEE
_____ DIVISION

KENDRIA Y. WEST

Name of Plaintiff(s)

v.

UNITED STATES OF AMERICA

Name of Defendant(s)

Case No. _____
(To be assigned by Clerk)

COMPLAINT

1. State the grounds for filing this case in Federal Court (include federal statutes and/or U. S. Constitutional provisions, if you know them):

Negligence, Fraud, Collusion, Abuse by Department of Veterans Affairs Medical Staff pursuant FTCA, sections 1346(b) and 2671-2680, title 28 USC, Intentional infliction of emotional, physical and financial distress

2. Plaintiff, KENDRIA Y. WEST resides at

1109 BLUEWILLOW CT, ANTIOCH,
Street address, City

DAVIDSON, TN, 37013, 615-641-1919,
County, State, Zip Code, Telephone Number

(If more than one plaintiff, provide the same information for each plaintiff below.)

3. Defendant, UNITES STATES GOVERNMENT resides at

3322 WEST END AVENUE SUITE 509, NASHVILLE,
Street address, City

DAVIDSON, TN, 37202, (615) 695-4633,
County, State, Zip Code, Telephone Number

(If more than one defendant, provide the same information for each defendant below.)

DEPARTMENT OF VETERAN AFFAIRS
OFFICE OF REGIONAL COUNSEL
810 VERMONT AVE
WASHINGTON DC 20420

4. Statement of claim. (State as briefly as possible, the facts of your case. Describe how each Defendant is involved. Include also the names of other persons involved, dates, and places. Be as specific as possible. You may use additional paper if necessary. Attach any documentation or exhibits in support of the complaint):

See Attached

5. Prayers for Relief (List what you want to Court to do):

a. Make it CRYSTAL CLEAR to the Department of Veteran Affair that MATTERS OF FACT MUST BE ACCURATLY DOCUMENT AS SUCH! How long a person had to live is a matter of OPINION. A diagnosis is a matter of FACT and is based on microscopic and laboratory testing NOT HE SAID, SHE SAID! Even with that testing must be COMPLETE AND THROUGH. There are manual and ESTABLISH protocol for assessing a patient, they don't just get to make it up as they go along.

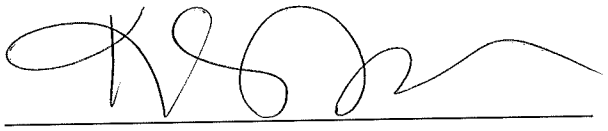
b. Make the offending physians provide me their full practicing name and license numbers so that I may report these attrocities along with my photographs and lab work to the Dermatological Society/Board as well as the Medical Society/Boards.

c. Redact my personal medical information after the case has ended. It is necessary to provided to establish the facts of this case for the Judge and Jury.

d. Award me generous compensation for my continued problems association with my ailments that the contractor maliciously refused to assess properly. At least three are service connected and can be shown to be as such via my medical records and the doctors appear to be told to lie so as not to justify compensation adjustments. I have advanced education but am unable to work due to exhaustion and other problems associated with my medical issues. Reimbursement for labs, microscope, and photo that they should have performed but did not.

I (We) hereby certify under penalty of perjury that the above Petition is true to the best of my (our) information, knowledge, and belief.

Signed this 02 day of November, 2017.



(Signature of Plaintiff(s))

On 05/20/15 I saw Dr Arzubiaga at the Alvin C York VA Medical Center for a case of trichorrhexis nodosa which photographs (***Exhibit A***) SHOW THAT I HAVE. She told me that "no I didn't have trichorrhexis nodosa" (***Exhibit B*** shows Google and The Manual of Dermatology definition and pictures which CLEARLY match my own hair samples) and that I should try things that no other dermatological professional was suggesting in published material. The exception was that of "stopping chemical processing" WHICH I HAD ALREADY DONE for several long periods over the years. She performed no testing AT ALL. So I immediately filed a SF95 (***Exhibit C pgs 1-5***) which describes why she was negligent and breached her fiduciary duty to provide me with a competent health analysis and actual health care. Accordingly, I was given a new appointment to correct the problems that I had with Dr Arzubiaga to see Dr Livingood on 09/17/15 at the Nashville VA Medical Center at which I was subjected to having fraudulent and/or deceptive statements made in my medical files which I will outline thoroughly later in this writing with additional exhibits. Dr Livingood took my hair that I brought in for evaluation and threw it into the trash IMMEDIATELY WITHOUT LOOKING AT

IT. He then called in Dr Sticklin to join the appointment and told me FLAT OUT that "they were not going to perform any testing and that they were going to collude and support the non-performance of Dr Arzubiaga" because "their OPINION mattered more because they are doctors". Dr Sticklin told me that "he didn't care about my filing the complaint because he was retiring and his career wouldn't be affected." Since the problem in the original SF95 was not corrected, but in FACT escalated in bad behavior, I filed an additional SF95 (***Exhibit C pgs 9-13***).

Dr Livingood is WRONG and it is FRAUD to write lies into person's medical records as a Medical Professional. So in that way the False Claims Act was also violated as he is being PAID to provide medical care as a contractor/employee but instead conspired and colluded to commit fraud, deception and abused me and my health by not doing a proper medical evaluation. Also, ALL DOCTORS need to know that patients including myself do not go to doctors for their "OPINION" but go so that the doctor can perform TESTING that provide additional FACTS to help with correct disease process identification. As I will later explain, symptoms can be attributed to any number of disease processes and a

medical professional is suppose to use TESTING to "rule out" one disease process versus another with "FACTS". In addition, they all caused me the stress and anguish of having to file a lawsuit and additional physical and psychological suffering from them not doing their jobs in the first place. Dr Sticklin was also complicit and nodding as Dr Livingood behaved defiantly and contrary to the purpose for which he was hired/contracted. Dr Arzubiaga DID NOT PERFORM HER JOB AS A PHYSICIAN, however, she did not state to my face that she was engaging in a fraudulent conspiracy the way that other two did but she did make entries in my medical record that seems to indicate her willing participation as well. That FACT will be illustrated in a later exhibit. In sum, they were negligent and the amount of emotional abuse and distress that I was subjected to was disturbing at every level! On 05/25/17 I was given permission to proceed with this filing because "a dermatology specialist stated that they'd done nothing wrong" (***Exhibit C pgs 14-16***). They didn't receive my permission to provide my medical information to any "dermatology specialist" so it seems that some HIPAA privacy violation has occurred as well.

A thorough account of the FACTS of this filing is what follows:

First, my background that qualifies me as an EXPERT to speak on my medical situation is that I was a Hospital Corpsman for 03/86 to 09/90 in the United States Navy. My duties included working in the hematology/oncology where I assisted with chemotherapy administration, medical record documentation, and patient education classes. Next was the Primary Care Clinic where I triaged patients, performed phlebotomy, picked or ordered labs and x-rays for diagnostic purposes. I also administered medicine via injections. On Okinawa I worked in pediatrics where I gave immunization and did hair shaft analysis to check for lice, assisted with spinal taps, circumcisions, and other medical procedures, ordered labs and x-rays for diagnostic purposes. I worked in the ER to perform emergency medical services, and in the Surgery Clinic where we performed Endoscopy, Bronchoscopy, Sigmoidoscopy, Colonoscopy and non-operating room surgical procedures under local anesthesia. The surgeons also worked in the operating rooms but I did NOT! I assisted with the follow-up check ups and removed staples and sutures. I was also a Nationally

Certified EMT. So through education and work experiences I COMPLETELY understand the day to day operations of Healthcare! As medical corps staff were are trained just a thoroughly as most physicians and taught to use the S.O.A.P. methodology which is detailed in ***Exhibit D*** primarily because MD's do NOT go to the field in war so we actually have to know what we are doing to save the lives of our service members until they are able to get to the hospital where MD's are kept. After leaving the service I received my Bachelor's and Master's Degrees in something else and though I no longer cared to be a healthcare worker I am still in possession of the knowledge and continue to update it through independent study.

Exhibit E shows that on 02/09/2015 I saw my primary care physician Dr Chakravarthy and asked for a consultation to Dermatology for "trichorrhexia" and "Hyperpigmentation" that he too noticed and documented as on my lower left lip, dorsal hands and knuckles. I have NEVER told any physician that I had "hair loss" and have NEVER complained of a "scalp problem" besides dandruff. ***Exhibit F*** shows that on 01/17/12 I requested shampoo for my dandruff which could have been cosmetic or medical and was given it without

question or hesitation from a VA Medical Professional. I knew that I had trichorrhexis nodosa because whilst standing outside in 1988 my pediatrics coworker wanted to check my hair for lice when she saw it in the sun because trichorrhexis nodosa 'looks like" lice nits until you examine it closely. We had to immediately report it to the Doctor's in pediatrics if we saw in a child's hair because it is indicative of urea cycle disorders that *typically* shows up in children and is quite dangerous. One of the doctors suggested that I make sure that it wasn't my styling choices and so I employed a beautician to assist me with hair care decisions but the Trichorrhexis Nodosa didn't respond. I was NOT formally seen for the problem so that is why I wouldn't expect and didn't have testing, but it was witnessed so I'm SURE that it occurred during military service. In addition, since High School I have been an avid vitamin taker so no vitamin deficiencies were suspected. In FACT one of the surgeons told me to "stop taking my vitamins because you just pee them out!" I kept taking them and still do. Then one day the trichorrhexis nodosa just stopped. It was off and on for years but I was prohibited from consulting a physician through my work

insurance due to the "preexisting condition" insurance clause. In 1996 the trichorrhexis nodosa was continuous and I was developing more health issues so I left work to see what was going on. At this point I discontinued relaxing my hair for several long periods to no avail. So I had ruled out abuse as a cause. As a matter of FACT when I removed my relaxer my breakage was worse. By 2003 I was totally physically depleted but due to my inability to work I had the ability to really dig deep into the books, my military medical record and medical scientific studies to address my health issues by researching for myself. During my research I discovered that trichorrhexis nodosa could be associated with thyroid problems which one of my coworker physicians thought that I had or would have because I had **extreme** temperature sensitivity. So I grinned and went up to the Murfreesboro VA Hospital and in 2003 my suspicions were confirmed as I had low T4 and normal TSH which is a completely odd presentation of hypothyroidism which would not have been found if the doctor had only checked the TSH which is what is typically done and is only indicative of primary hypothyroidism. I gladly took the synthroid but

noticed that my trichorrhexis nodosa only improved by about 50%.

In my *former* medical worker's mind that meant that something else was going on with my endocrine system. So I asked to see a specialists (dermatology and endocrine as they can overlap) which I eventually got to do. The endocrinologist was horrible and similar to the 3 doctors listed in this complaint. For example, he had me drink water whilst he touched my throat and then declared that "I did not have thyroid nodules" but when I had a subsequent ultrasound I was found to actually have at least 2. Things like that are what prompted my serious concerns about his abilities. I did file complaint with the VA but unfortunately did not yet know about this process so it is mentioned for information purposes only. It needs to be mentioned to establish the FACT that I am filing this complaint out of necessity as the VA did nothing. His testing was VERY minimal and questionable but I did learn that I had thyroid antibodies which totally meant that I had AUTOIMMUNE HYPOTHYROIDISM. Not an opinion but a FACT. I then learned that it is often associated with celiac disease. The celiac disease was mentioned to me 03/29/88

during an ER visit when I was stationed in San Diego. I had SEVERE right upper quadrant pain which the treating physician said was either afebrile appendicitis or celiac disease. He gave me a "GI cocktail" which relieved the pain. This meant that was most likely celiac disease and that I wouldn't have to undergo exploratory surgery because the pain resolved. In his notes (***Exhibit G pg1***) he told them that "if the pain persisted that they should consider an endoscopy". It was the only way that celiac disease was diagnosed back then. The pain DID persist (***Exhibit G pgs 2-3***) but they didn't test me as he indicated, but instead did a barium swallow test which obviously didn't show the problem. It was "a" test but not one that would actually identify the problem. Again since it was obviously present, preexisting and documented I could not use my work insurance for testing. The VA Murfreesboro had autoclave and sterilization problems where they infected patients with hepatitis and HIV so it wasn't an option to be tested there via endoscopy. The VA Nashville wasn't reporting the same type of issues but after seeing the a couple of physicians there who performed and tested contrary to reality I preferred to stick with the VA Murfreesboro

facility because physician performance was better but I wasn't risking invasive procedures at either of their facilities. By 2008 science had advanced and DNA testing for Celiac Disease was available and so with great financial difficulty, I had the testing done and it was found that I had 2 HLA-DQB1 genes indicative of celiac/gluten issues. Specifically I had 1 gene for celiac disease and 1 gene for gluten sensitivity! That is one from each parent, so I have a 0% of successfully eating gluten and my food is VERY expensive as a result. The gene work was actually performed by the American Red Cross for a lab that specializes in gastric issues so it is of respectable quality. I submitted the laboratory findings to the VA and Dr Jory S. Simmons Sr validated that FACT and his concurrence is shown in ***Exhibit H***. Please note that his writing was based on DNA TESTING and not on his OPINION! Not every doctor associated with the Department of Veteran Affairs behaves in a way that is dishonest and detrimental to patient care but I've encountered it more often than not particularly at the Nashville VA Hospital. DNA testing doesn't "fluctuate" as other testing does so this is a *legitimate* reason not to perform additional testing such as

an endoscopy. Endoscopy and Plasma results can fluctuate depending on whether or not gluten is eaten and I'd stopped as soon as I realized the link. Even with being gluten free the problem still shows its "presence" in the plasma testing. Also if you look at my work experience in the military I actually worked and assisted with those procedures when I worked in the surgery clinic, HOWEVER, in the military you MUST go to a set of "staff physicians" to get a consultation to have a procedure done. The "staff physicians" were content with "medicating the symptoms" instead of "discovering the problem" and as a result the problem was never correctly diagnosed but is document quite well.

In response to the appointment on 02/09/15, Dr Chakravarthy requested a **new consultation** for Dermatology and on 05/20/15 I saw Dr Arzubiaga who did absolutely NOTHING. First if you look at Dr Arzubiaga Progress Notes from 05/20/15 (***Exhibit I***) you will see the following:

- ***Exhibit I pg 1*** shows that this was a "MU-DERMATOLOGY NEW" consultation which means that she was suppose to be assessing this problem HERSELF!

- ***Exhibit I pg 2*** shows that she considered my problem to only be "cosmetic" when in actuality Trichorrhexis Nodosa is mostly medical. Poor or abusive hair care is the only non-medical cause of Trichorrhexis Nodosa and since it is in the "Medical Journals" it is considered by professionals as a MEDICAL PROBLEM not a "cosmetic problem". Biotin is a nutrient that helps hepatitis, brittle nails and hair, neuropathy, insulin resistance and depression. If she thought that I had a "biotin deficiency" she should have tested! I was already taking "b-right" b-complex for years so I knew that if it was deficient at that point I had an inherited biotin deficiency as described by ***Exhibit J*** which was going to need even more testing and so it needed to be FACTUALLY identified. She also should have not suggested "more protein" unless I was deficient! According to ***Exhibit K*** for my age and weight I only needed 53.1 grams a day! Too much protein can cause kidney problems and can also cause problems if I actually had late onset urea cycle issues. BOTH OF THESE THINGS NEEDED TO BE TESTED FOR BUT she did NOTHING. She also should have done hormone levels as

indicated in ***Exhibit L,*** but she did NOTHING! Again medical problems not cosmetic ones. A more correct term is that Trichorrhexis Nodosa is an "external symptom".

- ***Exhibit I pg 5*** show "PROCEDURE DONE: No" which is the truth. It also says that "NEW MEDS PRESCRIBED AND REVIEWED BY MD" which is untrue, she told me to buy some biotin!
- ***Exhibit I pg 6*** is where she started to go back in time and to make fraudulent inserts from a 2009 appointment to try to cover that fact that SHE ACTUALLY DID NOTHING THAT WOULD BE CONSIDERED PATIENT CARE! At the top of this page it says "OTHER NOTES: Veteran is to: stop hair relaxers, use hand lotion to condition hair rather than hair conditioner, incorporate 90 grams of protein into diet daily, return one morning for blood draw in the out-patient lab (cortisol level)." and this is where her "P" plan and notes ACTUALLY STOPPED! But when you look further down the page she writes that "She was seen in Nashville 2009" and she then copied the notes from that 2009 appointment to give the "appearance" that she'd actually documented the

information correctly but in reality if you look at the S.O.A.P. plan she was already on "P" on page 5! It is fraudulent to do this when documenting "your interaction with the patient". This is where she was suppose to be writing "S" which is "what the patient tells you" but she is in FACT just copy and pasting from an old appointment. Keep in mind that the 2009 notes state that I'd "cut my hair" to go natural so if she'd listened to me or read when she was copying and pasting old material she should have seen that discontinuing the relaxer HAD ALREADY BEEN TRIED BEFORE I SOUGHT HELP FROM A PHYSICIAN EVEN BACK THEN and HENCE SHE SHOULD NOT BE RECOMENDING IT AGAIN. Her suggestions were horrific and out of touch with medical thinking and behavior. She should know that even if it was "only cosmetic" that women suffer psychological damage from hair issues and that this damage can be just as devastating as any other illness. No other dermatologist or manual suggested that I not use a hair conditioner but hand lotion. I need COMPLETE endocrine testing because if I have adrenal issues it is related to the US NAVY forcing me

to redo the PPD which in known to cause adrenal issues (***Exhibit M***). ***In Exhibit M pg 1*** you clearly see that it says was a "18 Mar1986 PPD Converter - Do Not Give" which means that I was to NEVER AGAIN be given a PPD! But on 29 Jul 1987 I was subsequently "ordered" to retake the test! This reportedly causes adrenal problems and testing for these problems seem to not fall into the "just a cortisol test" category! Either Dermatology or Endocrinology could work up the adrenal issues; however I already knew that only a cortisol test was not going to identify the issue if it wasn't primary adrenal failure which is doubtful. Once she suggested the "lotion" for my hair I knew that her work up was probably going to not be thorough enough to identify that problem either.

- ***Exhibit I pg 7*** she got even crazier and from the 2009 appt put "7x7 cm patch", "no discrete areas of alopecia" and "Labs' Free Testosterone, DHEAS, 17-OH progesterone, prolactin, iron, TIBC, ferritin: WNL" WHEN CLEARLY THE ONLY LAB TEST THAT SHE WAS WILLING TO ORDER WAS THE CORTISOL! How could the labs be within normal limits when she didn't order any and what

person in their right mind would think that lab results from 2009 would still be valid in 2015? If they were accurate (they were not and I filed a complaint about it back then but only to the VA because I didn't know about this process) they still would have needed to be tested in 2015 because this was a NEW CONSULT and those are results that fluctuate! SHE FAILED to do any real assessment and didn't think to include the 2009 date next to the labs listings thereby giving the impression that she'd actually performed testing! It gets EVEN CRAZIER when you go to "Biopsy of scalp" which ***Exhibit I page 5*** already told you that she did NOT do. Then under "DIAGNOSIS": it says "MILD NON-CACATRICIAL ALOPECIA" when in the "EXAM" section on the ***Exhibit I page 7*** says "no discrete areas of alopecia"! YOU CAN SEE ALOPECIA, you don't need the microscope for that. The 2009 appointment information should have NEVER BEEN PASTED INTO HER APPOINTMENT EVALUATION and the only reason to have done so is an attempt to deceive! The 2009 appointment is also moot because it is not within the statute of limitation of the SF95 filings and was

not part of my complaint. In addition, I'd already filed complaints directly with the VA about the lies associated with the 2009 appointment and the inconsistencies are still evident in her copy paste job. If there was a complaint and I saw so many inconsistencies in the material as a "doctor" I most certainly wouldn't be copying and pasting from the information! With Trichorrhexis Nodosa THE HAIR GROWS but there is a problem with gaining length, it's not a bald spot!

- ***Exhibit I pg 8*** is where her notes finish again, and you see the only lab test that she ordered was the "cortisol am" which is only 1 of 3 reasons that hyperpigmentation occurs. What about the other things? What about non-primary adrenal problems?
- ***Exhibit I pg12*** you may notice that the date is "05/19/2015 09:39" and it is not uncommon for the staff to start preparing for the next days appointment. It is one of the only things in this exhibit that was not done deceptively.
- She did not correctly diagnosis the breakage as trichorrhexis nodosa. I absolutely DID bring in hair

samples of the breakage so that it could be correctly identified. This is NOT mentioned as it should have been. With correct identification of the ACTUAL PROBLEM she would be more capable of performing proper testing to get to the source of the problem.

- If you look as the "S" of her notes where she is suppose to write **what I tell her is happening** you will see only partial statements of what I told her. She has points of what I said but she TOTALLY LEAVES OUT AND IGNORES THAT I'D ALREADY RULED OUT ABUSE AND WAS RECOMMENDING IT AGAIN INSTEAD OF MOVING ON TO ADDITIONAL TESTS which is why I was seeking professional help as suggested in ***Exhibit J pg 2*** where it says "When to contact a Medical Professional". I also did look around to see if there were any references of relaxers actually helping the condition of the hair like it did for me and on page 54 of The Manual of Dermatology hair disorders section there is such a reference. (***Exhibit N***)
- After hair abuse is ruled out more testing needs to be done so I went to ***Exhibit L*** and had the "Diagnostic Tests" performed with the exclusion of the scalp

biopsy, hair pull, and densitometry because the latter 3 deal with hair loss and not hair breakage. I also went through the "Causes" in ***Exhibit J pg 1*** because that would add vitamin testing, and urea cycle testing which is a major and dangerous cause of trichorrhexis nodosa.

After all of the things that I knew should have been done that were not done; I filed the first SF95 (***Exhibit C pg 1***). This gave the Department of Veterans Affairs the ability to look and to see that nothing was actually done and to actually perform the needed tests to identify my problems.

So on 09/17/15 I was scheduled to see Dr Livingood and ***Exhibit O*** shows the Progress Notes from that appointment.

- In ***Exhibit O Page 1*** he is listing my "Chief Complaint: f/u hair loss". THIS IS A LIE. I requested to see them for "Hair Breakage" and this lie is what led to the 2009 conflict that is not part of case and it was not what he was suppose to be doing. He was supposed to be evaluating me for "hair breakage" because Dr Arzubiaga failed to do so

in her 05/20/15 appointment which was to be a NEW CONSULTATION. The 2009 appointment is also moot because it is not within the statute of limitation of the SF95 filings. In addition, I'd already filed complaints directly with the VA about the lies associated with the 2009 appointment. I went there for trichorrhexis nodosa and they asked me if they could "just do the biopsy to gain clarity" but they wrote lies. I went to the Hair Club for Women IMMEDIATELY after getting the biopsy report and the technician performed a microscopy scalp examination for free and said that other customers "wished that they had that hair ratio that I do and that their physicians wouldn't even see me but that I could buy extra conditioning shampoo and conditioner product" which I refused to do! I endured months, and months, and months of harassing sales calls because of that biopsy lie. I am not in the market for hundred dollar shampoo no matter what! I have curly hair and so as it grows in, it groups into cowlicks so it can look "clumped" but I don't have alopecia according to the hair experts of the Hair Club for Women who

make MILLIONS addressing such issues! As such the 2009 appointment and details are incompetent, irrelevant, and immaterial to either of the sf95's (***Exhibit C***) that are part of this submission. He also notes that I was "adamant that metabolic or hormonal abnormalities are the likely cause" but he fails to test it because of some fake things that they wrote in the 2009 appointment! Of course I was ADAMANT because alopecia is cause by autoimmune factors and FOR SURE I have autoimmune thyroiditis and celiac BUT I WAS NOT EXPERIENCING hair loss per se but hair breakage! I keep my protein level optimal and have ALWAYS taken vitamins so that was something that I didn't need to try, it has always been. He does acknowledge the fact that I have hair that I "would like examined under a microscope", but leaves out the fact that he IMMEDIATELY threw it away and bullied me!

- In ***Exhibit O pg 2*** Dr Livingood is now also describing the spot as "4x4 cm of breakage" whereas Dr Arzubiaga is cutting and pasting a "7 x 7 cm" value! He then writes that the "remainder of scalp

w/o focal or diffuse hair loss" when the irrelevant scalp biopsy occurred half way my head in the parietal area so if there is "no focal or diffuse hair loss on the remainder of the scalp" then I CAN'T POSSIBLY HAVE ALOPECIA! The 4 x 4 spot was in the temple area and not in a place where a scalp biopsy would be safe! My head, as will be see in the court is the best evidence of the FACT that I don't have alopecia and I only mention details of the 2009 appointment to give clarity of the collusive and manipulative tendencies of these 3 individuals and what I endured at the VA Nashville facility in general. The whopper of his FRAUD is ***Exhibit O pg 2*** where he states that "no irregularities of the hair under light microscopy today on exam" when he didn't use a microscope on my head, AND ACTUALLY threw my hair samples in the trash as soon as I gave them to him. I still have the ziplock bag of with some of the trichorrhexis nodosa hair from that day that I have photographed in ***Exhibit A 1-20*** and can bring to court show that he is telling a lie! Yes, I got a microscope with a camera FOR PROOF of his lies! I

actually have no idea who was suppose to have done the FSH and LH that he's listed but an independent lab shows THAT IS A LIE TOO, and the fact of the matter is that HE WAS SUPPOSE TO BE DOING IT! If it was endocrinology it will be dealt with separately. The values associated with that endocrinology department were always off! FOR YEARS they told me that my prolactin was elevated and an independent laboratory reports that it is not. That endocrinologist could NOT tell me a "cause" or how to reduce it but was always reporting it. In addition, I have fibroids which are associated with progesterone deficiency according almost all quality medical professionals in the field, so based on that fact I immediately told them the 09/09/09 labs were off. Then, I couldn't afford to have the tests performed independently to "show" it and that is what they were counting on with these antics but, SURPRISE, I had it done this time! He then goes on to say that I have "non-scarring alopecia of unknown etiology: most likely due to trauma of traction;" when he'd previously and accurately described the

problem as "mid-hair breakage"! Then there is that "Lab workup in the past has ruled out sufficient causative nutritional causes" when I've have minimal vitamin testing done at their facilities and none of it included a "biotin" test which is what Dr Arzubiaga was demanding that I buy and take! In addition, notice that he's recommending that I should use "regular conditioner and mild shampoo" and not "hand lotion" and why is he suggesting for me to "stop chemical relaxers" if my problems is due to "trauma or traction" which involve "pulling"?! In addition, all of the past testing is MOOT BECAUSE THIS WAS A NEW CONSULTATION AND WAS SUPPOSE TO BE A NEW EVALUATION AND BOTH DOCTORS FAILED!

- In ***Exhibit O pg 3*** Dr Livingood states that I was "combative during clinic visit and appeared fixated on thought that there is a metabolic/systemic cause responsible for her focal hair loss." I was actually FURIOUS at their bullying and the tossing of my hair and the fact that he DID ABSOLUTELY NOTHING TO ACTUALLY ASSESS MY HEALTH! He was NOT strangled with his stethoscope or socked in the eye, so I was NOT

combative! Dr Livingood then said she "believes that she has trichorrhexis nodosa as well given her personal reading". Actually it was due to my training in pediatrics, microscopic pictures from ***EXHIBIT A*** and the matching of my ACTUAL MICROSCOPIC PICTURES to all existing literature on the subject! He is right I was "unwilling to listen to hypothesis that focal hair loss could be due to traction and manipulation" BECAUSE I WAS EXPERIENCING "mid-hair breakage" not traction or manipulation causatives!

- In ***Exhibit O pg3*** as promised in the bullying session Dr Stricklin just wrote that it was "not consistent with and endocrine cause". Which turned out to be a LIE and since he didn't correctly supervise his employees and IN FACT ENCORAGED THEM TO COLLUDE AND COMMITE FRAUD HE IS JUST AS GUILTY AS IF HE WERE THE ORIGINAL CONSULTING PHYSICIAN.
- The diagnosis of Trichorrhexis Nodosa was not correctly made by the second chance physician or his supervisor. Dr Livingood COMPLETELY IGNORED THE HYPERPIGMENATION ISSUE which my primary doctor saw

and documented. It fluctuates but he didn't even looks as shown in ***Exhibit O***.

So after the 09/17/15 appointment I immediately filed the additional SF95 (Exhibit C pg 6) and started to have the medical testing that Dermatology should have done instead, bullying me and committing fraudulent acts.

I was even more FURIOUS when I discovered that I had to counter the "cut and paste" of old information progress reports from Dr Arzubiaga and Dr Livingood with FACTS so I began to order the testing that competent Medical Professionals said would identify the problem. ***Exhibit L*** shows the testing list that I used but I also added Urea Cycle testing because it is a known cause of Trichorrhexis Nodosa. There is also Adult Onset Urea Cycle Disorders as described by ***Exhibit P*** so that was tested for as well. ***Exhibit Q*** shows that on 09/25/15 I had an Amino Acid Plasma test and a Ceruloplasmin test. The Amino Acid Plasma test was to highlight any Urea Cycle problems and the Ceruloplasmin test was to see if my skin darkening was associated with Wilson's disease. My alpha-aminobutyrate in the amino acids was slightly elevated. Problems associated with this include drinking alcohol, which I

never do and intestinal dysbiosis which is a possibility since I suffered so long with the gluten issues WITHOUT BEING PROPERLY DIAGNOSED!

Exhibit R shows that on 09/25/15 I had an Ammonia Plasma test that was shown to be in the normal range.

Exhibit S shows that on 09/25/15 I had FeGGT LifeProPremier performed. It is the checks for the irons associated with Trichorrhexis Nodosa but also checks for Hemachromatosis which is one of 3 things that could have been causing my discoloration. My MCV was high and the only associated disease it absolutely couldn't be was excessive alcohol intake as I've NEVER been drinker not even socially.

Exhibit T shows that on 09/28/15 I had a Functional Micronutrient test which shows that the only vitamin and mineral deficiencies that I have when on my daily supplementation plan are Vitamin B2, Vitamin D3 and Vitamin K2! I do NOT have a biotin deficiency and if Dr Arzubiaga or Dr Livingood had only TESTED before demanding that I go out and buy and try some biotin they would have known that FACT!

Exhibit U shows that on 12/19/2015 I had thyroid and adrenal testing which includes cortisol. The thyroid

antibodies are both above 0 but below 10 for the Anti-TPO Ab and detected but below 20 for the Anti-Thyroglobulin Ab. According to specialists that study this subject in medicine my thyroid is still under attack. Recent thyroid ultrasounds seem to confirm this FACT as I have "nodules" forming, but otherwise all values were in the normal range. I will add that because my adrenal are as likely as not to have been caused by the forced PPD shown in ***Exhibit M*** so it is unlikely that a cortisol would show the problem. So yes it was "a" test but not the right one, so it wasn't going to provide any additional information. I was expecting Dr Arzubiaga to perhaps perform response testing but she was not offering complete testing for that problem. You have to be injected to get response testing so it has to be performed in the doctor's office or in conjunction with a doctor's office and not just a lab.

Exhibit V shows that on 12/19/15 I had the suggested hormone balance profile performed. It was there that I found 3 problems. My follicle stimulation hormone FSH was high, my free testosterone was low, and my progesterone was low! Of those the only one that I'd be able to supplement was the progesterone. Within 3 months of starting

supplementation of the progesterone my Trichorrhexis Nodosa resolved and has remained that way so long as I supplement. This is PROOF POSITIVE that my 2009 complaint against them about "faked" lab work and biopsy results had basis. I was unable to borrow money to get testing done back then so I couldn't have come to court with my "opinion" but this time I borrowed from everybody that I knew and was able to SHOW THE FACTS and had learned about the SF95 process and am hopefully able to get JUSTICE! KEEP IN MIND that NO this does NOT fix my issues because the thyroid and progesterone showing deficiencies when not supplemented STRONGLY SUGGESTS that further problems lie in some failure that is occurring in the endocrine system overall and the EXACT PROBLEM still needs to be found! If ONLY EITHER of them had done their jobs the process of digging deeper to identify the disruptor of the system would have been started 2 whole years ago for the purposes of this complaint and in 2003 for the purposes of my quality of life! Because of all of the financial borrowing and paying back that I have to do I was only able to start progesterone replacement in 02/17 and so prometrium could have been prescribed a whole 2 years earlier if only they'd done their jobs.

Exhibit W shows that on 01/11/16 I had a 24 hour Urine Amino Acids performed. You will notice that some of the vitamins are lower that on the micronutrient test. This is because Genova Diagnostics requires 4 or more days with no vitamin or amino acid supplementation prior to their test. Hence it shows what my levels look like if I didn't take any supplements.

In sum, the evidence submitted shows the following:

1) That I ACTUALLY have Trichorrhexis Nodosa. (Exhibit A)
2) That I sought Medical Care for these problems from the Department of Veterans Affairs Medical System because they ACTUALLY occurred during my military service. I was unable to get outside help initially because law prohibited "preexisting conditions" from being seen by my work insurance plan. After the law was changed I was too sick to be able to afford insurance because the Department of Veteran Affairs has FAILED to pay me for the Celiac Disease and Thyroid Problems even though they'd began during my service and were documented in my medical records. They also did NOT fulfill their duty to assist in identifying these problems as laws states that they must. I was unable

to work because the ongoing problems give me SEVERE FATIGUE and due to sheisty behavior many things still have not been diagnosed correctly. If they initially fail to correctly diagnose a problem in the US Navy and the Department of Veteran Affairs fails in the "duty to assist" then the problems never get correctly document and Veterans physically suffer and are cheated for years and years out of fair and deserved compensation. For sure my Celiac Disease and Thyroid Problems are autoimmune in variety and that makes the US Navy a highly probably stimulator of these conditions with immunizations as they are a known trigger of autoimmune disease. The knowledge of this potential is fairly recent so I am NOT saying that it was done on purpose. In addition, EVEN IF I were able to afford insurance for "preexisting conditions" I cannot be guaranteed to have dealings with competent Physicians and this case is a stark realization of that FACT! Hence I sought medical help from the Department of Veteran Affairs NOT simply because I am a Veteran but because these problems were actually

beginning and documented WHEN I WAS IN THE UNITED STATES NAVY!

3) Had Dr Arzubiaga or Dr Livingood been taught or required to perform proper patient assessment and evaluation my problem would have been identified and actually resolved with proper treatment and the true process to root out the real culprit of my issues could have been pursed. Instead they lied, copied and pasted, abused me mentally, physically and financially and I've suffered greatly because of it as other conditions of my progesterone deficiency worsened.

4) Dr Stricklin is just as guilty and it seems that he may be the Department Head and instead of correcting the situation as he should have, he joined in on the abuse. I think that the attitude was that "she's poor because we haven't given her the money to which she is entitled to so she can't afford a doctor and even if she can he might not be good". Fortunately, I too know how to treat and manage medical issues and I also found a court that doesn't go on "cut and paste he said/she said" but on FACTS!

5) Progesterone deficiency is a KNOWN factor of female related cancers and other related disturbances some of which I have been shown to have. ***Exhibit X*** has an excellent presentation on the cancer aspects of this deficiency. ***Exhibit Y*** shows some of the other medical issues associated with progesterone deficiency. Though I suffer from other problems associated with this disorder that are not trichorrhexis nodosa NONE OF THEM tested me for this abnormality! IT IS KNOWN that progesterone deficiency causes cancer and fibroids and instead of testing for the appropriate deficiencies they try to expose you to radiation via mammography and try to con you into hysterectomies INSTEAD OF ACTUALLY PERFORMING PREVENTATIVE MEDICINE IN THE FORM OF READILY AVAILABLE TESTING and deficiency replacement! Lastly, ***Exhibit Z*** shows more detrimental effects of low progesterone and how MY PHYSICAL HEALTH was being affected not just my hair!

6) It appears that the basis for my "fake" 2009 results is because they don't want to pay my rightful compensation of 100%. If this has happened to me then it is happening to other Veterans; MAKE THEM STOP! If

they have a "duty to assist" then the assistance should not be full of fraud and corruption which not only affect the veteran's health but their finances.

7) That I must have substantial funds to have my health properly assessed. The conditions which started appearing when I was very young are now risking my life and greatly compromised the last few years of my fertility. Hence, I need to be able to seek competent medical assistance which these designated Medical Facilities have demonstrated that they don't know how to perform or are unwilling to perform.

8) This is happening because these doctors are being encouraged to "guess" and "not test" and it is VERY detrimental to a patients health and as more patients catch on it will be VERY detrimental to "doctors" that do this careers! It boils down to the fact that I am being given appointments BUT NOT ACTUALLY RECEIVING COMPETANT MEDICAL EVALUATIONS just like with the TUSKEEGEE EXPERIMENT. Another consequence of them not accurately and thoroughly testing is that they'd give me a $12,000 hysterectomy and a lifetime supply of estrogen and progesterone replacement but NOT a $200

hormone test and a $30 progesterone (prometrium) prescription and further evaluation to actually resolve the endocrine issues! They are FACTUALLY mutilating Veteran Women for profit!

9) Because it is MOOT I will not fully investigate the 2009 Dermatology Appointment but it WAS FRAUGHT WITH PROBLEMS WHICH WERE REPORTED TO THE VA. Then I briefly looked back and can actually SHOW that I first asked to see dermatology in 2005! I saw a Dr Fuchs who seems to have been correctly assessing my problem as I'd ALWAYS REPORTED IT AS "hair breakage". Dr Sticklin gets involved and all of a "sudden a letter was mailed but I didn't show" and my problems were being reported as alopecia and not breakage as it should have been. ***Exhibit AA*** shows these FACTS! I was perhaps up for review for my compensation and by not correctly reporting the Trichorrhexis Nodosa he was working against me to get it denied which means that he NEVER fulfilled his "duty to assist" obligations and they use the 2009 appointment information because that is when he could get the consulting physician to write lies! How'd I get a consult request in 2005 but not

get seen until 2009? It "appears" that he was up to no good from day one!

10) I've discovered a direct link between Progesterone Deficiency laboratory results and Trichorrhexis Nodosa which the "doctor" refused to look for and hence didn't find it!

11) This is DEFINITELY a case of intentional infliction of physical, mental and financial distress!

I would like to apologize to the court in advance for the length but this situation required that I THOROUGHLY go through why I am actually forced to file this case and why the Department of Veteran Affairs is responsible. It's not "just because I am a Veteran" but because these thing ACTUALLY HAPPENED DURING MY MILITARY SERVICE and it was due to ***THEIR*** failing that the conditions were not correctly diagnosed. In addition, I have significant fatigue issues so it was better for me to comb through thousands of pages beforehand to answer any potential questions that I anticipate and have the documents already available as ***exhibits*** for review.

www.google.com/search

trichorrhexis nodosa

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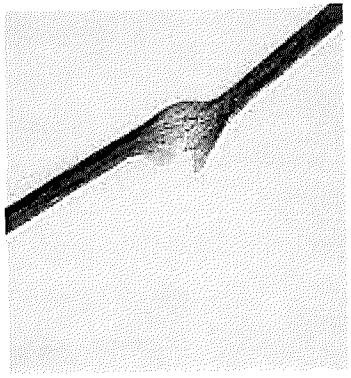
Trichorrhexis nodosa is a defect in the hair shaft characterized by thickening or weak points (nodes) that cause the hair to break off easily. This group of conditions contributes to the appearance of hair loss, lack of growth, and damaged-looking hair.



Trichorrhexis nodosa - Wikipedia
https://en.wikipedia.org/wiki/Trichorrhexis_nodosa

About this result Feedback

Exhibit B

- Clinical – most patients are blond girls (older than 2) who never need a haircut. May persist into adulthood. Hair is not typically brittle and of normal strength

- **Mitochondrial disorders**
  - May see trichorrhexis nodosa, trichorrhexis, longitudinal grooving, trichoschisis, pili torti
- **Marie Unna Hypotrichosis** – AD
  - Have normal to coarse sparse hair and eyebrows
  - Develop coarsening within first few years
  - Eyebrows, eyelashes, axillary hair also affected
  - Scalp – hair loss starts in parietal and vertex. Partial sparing of occipital scalp

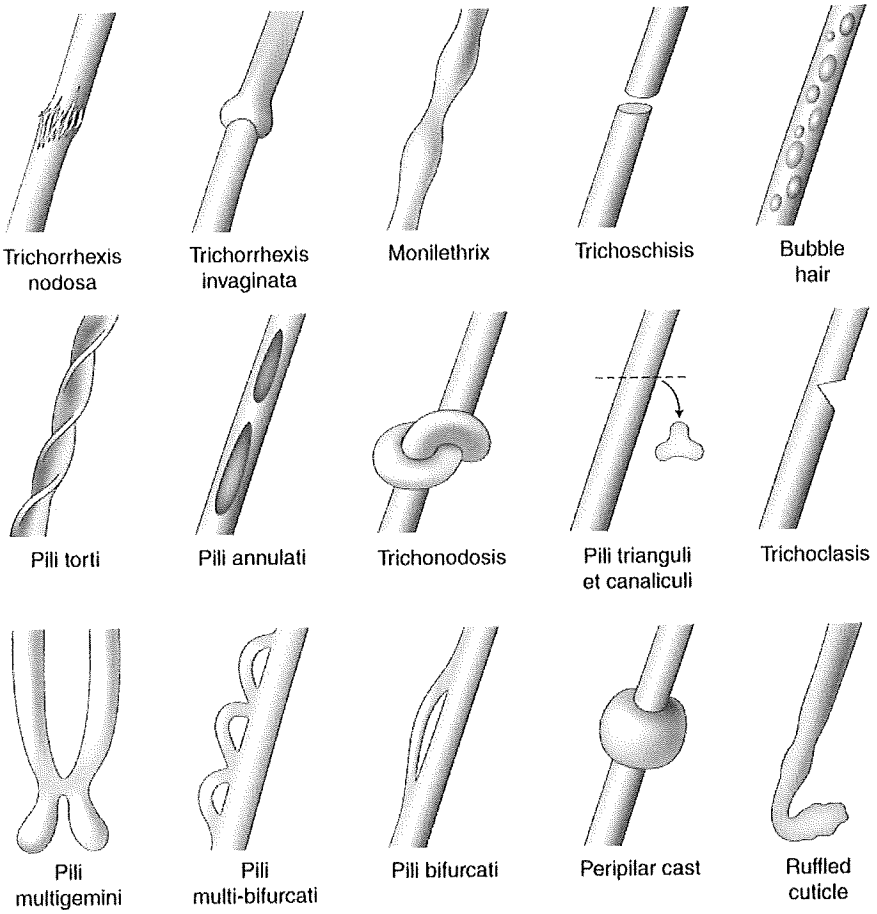


Fig. 1 Hair shaft anomalies

Exhibit C

CLAIM FOR DAMAGE, INJURY, OR DEATH

INSTRUCTIONS: Please read carefully the instructions on the reverse side and supply information requested on both sides of this form. Use additional sheet(s) if necessary. See reverse side for additional instructions.

FORM APPROVED
OMB NO. 1105-0008

1. Submit to Appropriate Federal Agency:

DEPARTMENT OF VETERAN AFFAIRS
OFFICE OF REGIONAL COUNSEL
3322 WEST END AVE, SUITE 509
NASHVILLE TN

2. Name, address of claimant, and claimant's personal representative if any. (See instructions on reverse). Number, Street, City, State and Zip code.

KENDRIA Y WEST
1109 BLUEWILLOW CT
ANTIOCH TN 37013

3. TYPE OF EMPLOYMENT	4. DATE OF BIRTH	5. MARITAL STATUS	6. DATE AND DAY OF ACCIDENT	7. TIME (A.M. OR P.M.)
[X] MILITARY [ ] CIVILIAN	10/05/1967	SINGLE	05/20/2015 WED	2:00 P.M.

8. BASIS OF CLAIM (State in detail the known facts and circumstances attending the damage, injury, or death, identifying persons and property involved, the place of occurrence and the cause thereof. Use additional pages if necessary).

SEE ATTACHED

9. **PROPERTY DAMAGE**

NAME AND ADDRESS OF OWNER, IF OTHER THAN CLAIMANT (Number, Street, City, State, and Zip Code).

N/A

BRIEFLY DESCRIBE THE PROPERTY, NATURE AND EXTENT OF THE DAMAGE AND THE LOCATION OF WHERE THE PROPERTY MAY BE INSPECTED. (See instructions on reverse side).

N/A

10. **PERSONAL INJURY/WRONGFUL DEATH**

STATE THE NATURE AND EXTENT OF EACH INJURY OR CAUSE OF DEATH, WHICH FORMS THE BASIS OF THE CLAIM. IF OTHER THAN CLAIMANT, STATE THE NAME OF THE INJURED PERSON OR DECEDENT.

Continued perforation of hair that IS NOT associated with relaxing as all of my hair does it. Continued emotional and psychological anguish from my hair breakage. Continued deterioration of my physical health from the underlying deficiency not being properly identified or addressed!

11. **WITNESSES**

NAME	ADDRESS (Number, Street, City, State, and Zip Code)
JEANETTEWEST	1109 BLUEWILLOW CT ANTIOCH TN 37013

12. (See instructions on reverse). **AMOUNT OF CLAIM** (in dollars)

12a. PROPERTY DAMAGE	12b. PERSONAL INJURY	12c. WRONGFUL DEATH	12d. TOTAL (Failure to specify may cause forfeiture of your rights).
0.00	1000000	0.00	1000000

I CERTIFY THAT THE AMOUNT OF CLAIM COVERS ONLY DAMAGES AND INJURIES CAUSED BY THE INCIDENT ABOVE AND AGREE TO ACCEPT SAID AMOUNT IN FULL SATISFACTION AND FINAL SETTLEMENT OF THIS CLAIM.

13a. SIGNATURE OF CLAIMANT (See instructions on reverse side).	13b. PHONE NUMBER OF PERSON SIGNING FORM	14. DATE OF SIGNATURE
	615-641-1919	06/05/2015

CIVIL PENALTY FOR PRESENTING FRAUDULENT CLAIM

The claimant is liable to the United States Government for a civil penalty of not less than $5,000 and not more than $10,000, plus 3 times the amount of damages sustained by the Government. (See 31 U.S.C. 3729).

CRIMINAL PENALTY FOR PRESENTING FRAUDULENT CLAIM OR MAKING FALSE STATEMENTS

Fine, imprisonment, or both. (See 18 U.S.C. 287, 1001.)

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NSN 7540-00-634-4046

STANDARD FORM 95 (REV. 2/2007)
PRESCRIBED BY DEPT. OF JUSTICE
28 CFR 14.2

95-109

INSURANCE COVERAGE

In order that subrogation claims may be adjudicated, it is essential that the claimant provide the following information regarding the insurance coverage of the vehicle or property.

15. Do you carry accident Insurance? ☐ Yes If yes, give name and address of insurance company (Number, Street, City, State, and Zip Code) and policy number. ☒ No

N/A

16. Have you filed a claim with your insurance carrier in this instance, and if so, is it full coverage or deductible? ☐ Yes ☒ No

N/A

17. If deductible, state amount.

0.00

18. If a claim has been filed with your carrier, what action has your insurer taken or proposed to take with reference to your claim? (It is necessary that you ascertain these facts).

N/A

19. Do you carry public liability and property damage insurance? ☐ Yes If yes, give name and address of insurance carrier (Number, Street, City, State, and Zip Code). ☒ No

N/A

INSTRUCTIONS

Claims presented under the Federal Tort Claims Act should be submitted directly to the "appropriate Federal agency" whose employee(s) was involved in the incident. If the incident involves more than one claimant, each claimant should submit a separate claim form.

Complete all items - Insert the word NONE where applicable.

A CLAIM SHALL BE DEEMED TO HAVE BEEN PRESENTED WHEN A FEDERAL AGENCY RECEIVES FROM A CLAIMANT, HIS DULY AUTHORIZED AGENT, OR LEGAL REPRESENTATIVE, AN EXECUTED STANDARD FORM 95 OR OTHER WRITTEN NOTIFICATION OF AN INCIDENT, ACCOMPANIED BY A CLAIM FOR MONEY DAMAGES IN A **SUM CERTAIN** FOR INJURY TO OR LOSS OF PROPERTY, PERSONAL INJURY, OR DEATH ALLEGED TO HAVE OCCURRED BY REASON OF THE INCIDENT. THE CLAIM MUST BE PRESENTED TO THE APPROPRIATE FEDERAL AGENCY WITHIN **TWO YEARS** AFTER THE CLAIM ACCRUES.

Failure to completely execute this form or to supply the requested material within two years from the date the claim accrued may render your claim invalid. A claim is deemed presented when it is received by the appropriate agency, not when it is mailed.

If instruction is needed in completing this form, the agency listed in item #1 on the reverse side may be contacted. Complete regulations pertaining to claims asserted under the Federal Tort Claims Act can be found in Title 28, Code of Federal Regulations, Part 14. Many agencies have published supplementing regulations. If more than one agency is involved, please state each agency.

The claim may be filled by a duly authorized agent or other legal representative, provided evidence satisfactory to the Government is submitted with the claim establishing express authority to act for the claimant. A claim presented by an agent or legal representative must be presented in the name of the claimant. If the claim is signed by the agent or legal representative, it must show the title or legal capacity of the person signing and be accompanied by evidence of his/her authority to present a claim on behalf of the claimant as agent, executor, administrator, parent, guardian or other representative.

If claimant intends to file for both personal injury and property damage, the amount for each must be shown in item number 12 of this form.

The amount claimed should be substantiated by competent evidence as follows:

(a) In support of the claim for personal injury or death, the claimant should submit a written report by the attending physician, showing the nature and extent of the injury, the nature and extent of treatment, the degree of permanent disability, if any, the prognosis, and the period of hospitalization, or incapacitation, attaching itemized bills for medical, hospital, or burial expenses actually incurred.

(b) In support of claims for damage to property, which has been or can be economically repaired, the claimant should submit at least two itemized signed statements or estimates by reliable, disinterested concerns, or, if payment has been made, the itemized signed receipts evidencing payment.

(c) In support of claims for damage to property which is not economically repairable, or if the property is lost or destroyed, the claimant should submit statements as to the original cost of the property, the date of purchase, and the value of the property, both before and after the accident. Such statements should be by disinterested competent persons, preferably reputable dealers or officials familiar with the type of property damaged, or by two or more competitive bidders, and should be certified as being just and correct.

(d) **Failure to specify a sum certain will render your claim invalid and may result in forfeiture of your rights.**

PRIVACY ACT NOTICE

This Notice is provided in accordance with the Privacy Act, 5 U.S.C. 552a(e)(3), and concerns the information requested in the letter to which this Notice is attached.

A. *Authority:* The requested information is solicited pursuant to one or more of the following: 5 U.S.C. 301, 28 U.S.C. 501 et seq., 28 U.S.C. 2671 et seq., 28 C.F.R. Part 14.

B. *Principal Purpose:* The information requested is to be used in evaluating claims.

C. *Routine Use:* See the Notices of Systems of Records for the agency to whom you are submitting this form for this information.

D. *Effect of Failure to Respond:* Disclosure is voluntary. However, failure to supply the requested information or to execute the form may render your claim "invalid."

PAPERWORK REDUCTION ACT NOTICE

This notice is solely for the purpose of the Paperwork Reduction Act, 44 U.S.C. 3501. Public reporting burden for this collection of information is estimated to average 6 hours per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to the Director, Torts Branch, Attention: Paperwork Reduction Staff, Civil Division, U.S. Department of Justice, Washington, DC 20530 or to the Office of Management and Budget. Do not mail completed form(s) to these addresses.

KENDRIA Y WEST
SF 95
BASIS OF CLAIM

On 05/20/15 I had an appointment to see the Dermatologist at the Alvin C York VA Medical Facility. She (Carmen Arzubiaga) didn't tell me her name, refused to look at my hair under the microscope and put her finger over the injured area whilst pretending to look at it under the big magnifying glass! The magnifying glass DOES NOT zero in like the microscope. Because of this, she failed to properly diagnose my hair shaft defect. My hair is perforating in spots so it's not a split ends issue! Perforations MUST BE a hair shaft defect but there are several that could be indicated. There is Bubble Hair, Beaded Hair/Monliethrix, Pili torti, Trichorrhexis invaginata, Trichorrhexis nodosa, or Trichothiodystrophy! She needed to use to microscope to get this correct and she didn't!

She told me to "try" some biotin and that she'd put in for it but didn't know if it would be approved. In addition, she told me that Biotin was NOT listed as a B vitamin! It IS listed as B7! I have attached the instructions that I was given at this appointment. This is usually typed into the computer and printed out but it wasn't this time. She also stated that I should "use lotion" instead of using harsh conditioners. Her advice is NOT sound considering that I use a natural baby shampoo and conditioner, don't relax my body hair but have the same problem there as well. In addition, high protein diets are taxing on the kidneys and she performed not one test that shows that I'm protein or biotin deficient! Just so that you understand that I understand a Biotin Deficiency workup consists of **biotin levels**, **serum ammonia levels**, **urine ketones levels**, **quantitative plasma amino acid levels**, **plasma carnitine levels**; **both free and total (sometimes termed free and esterified)** , and a **routine serum chemistry panel**! As such, it's NEVER really the doctor that determines what's wrong it's the TESTS which she failed to run!

Doctors are to properly evaluate patients to arrive at a proper and accurate DIAGNOSIS. Doctors are IN FACT scientists that use science to determine health problems. It is NOT APPROPRIATE and DANGEROUS to patient health to have patients to "try this and that" without properly identifying the problem. It is tantamount to malpractice because my problem still exists and causes not only psychological and emotional trauma but the further deterioration of the actual health deficiency! If you pay attention to the fact that she suggested a biotin deficiency and that work up includes serum ammonia level and quantitative plasma amino acid levels and you know anything you'd recognize that hair breakage like mine is characteristic of a urea cycle defect (which is curable but deadly) and the protocol testing which she failed to perform is seeking to find the actual deficit so that it can be corrected. Urea cycle defects are typically seen in children (I worked in pediatrics and I've seen a case of a 2 year old with one which is why I sought help) HOWEVER, it can occur in adults where it is termed "late onset" and triggered by illness! How long a patient will live is a matter of opinion, whether or not a patient is deficient in this, that or the other is a

Exhibit C

KENDRIA Y WEST
SF 95
BASIS OF CLAIM

MATTER OF FACT! So this is a FACTUAL case based on failure to perform her duty!

In addition, I demonstrate financial need but her request for the VA to supply my biotin was DENIED because she failed to demonstrate the need by performing appropriate and necessary tests! She only offered a cortisol test to see if it was the cause of my hyperpigmentation which is still wrong for that as well. Cortisol is best as saliva but can be serum or urine. In addition, the whole endocrine system would need to be evaluated; also cortisol fluctuates so there is am/pm and 24 hour. So she did nothing correct during my visit.

Based on this experience Dr Arzubiaga has failed me miserably and put my emotional, psychological AND physical well being into continued jeopardy! It is this FACT that is the basis for this form submission.

After I initially submitted a SF-95 regarding this I was re-seen (as I was to allow in an attempt to resolve this issue) on 07/17/16 by the Dermatology Clinic at the Nashville Facility. During this visit I saw a Dr. Livingood who proceeded to tell me that though she'd done NOTHING that "Dr Arzubiag had done a good job." and that "since I didn't have a job I wouldn't be able to get testing done to show that she didn't." He went on to go get Dr. Stricklin who told me FLAT OUT that he was going to "say the same thing because he didn't care if I refilled because he had his 30 years and was retiring." So though I allow them to correct the problem they chose to try to intimidate me and DID NOTHING!

In light of that situation I prepared to refilled and prepared to show THE INTENTIONAL AND MALICIOUS FAILURE of the Dermatology Department of BOTH FACILITIES in the Nashville Area. This continued failure made it essential for me to re-file, increase the requested damages, and SHOW that they were wrong!

I did receive notice from Tammy Kennedy that she was denying the initial request which was fine with me because of the progression of the problem. However, in her letter she stated the "an outside physician said that nothing wrong was done." This COULD HARDLY BE TRUE because is NEVER not wrong to properly evaluate your patient. In addition, I NEVER SIGNED ANY RELEASE OF INFORMATION for ANY OTHER DOCTOR to review my information, so in doing so she violated my privacy rights. In addition, trichorrhexis nodosa is a visual diagnosis and he most certainly didn't see me or my hair so it would be impossible for him to know if she was wrong or not!

So with those facts now documented, I also submit CLIA certified laboratory results and ACTUAL PICTURES OF MY HAIR PROBLEM so that as this case proceeds the judge and jury SEE that I'm NOT participating in ANY case of he

KENDRIA Y WEST
SF 95
BASIS OF CLAIM
said, she said but am dealing with objective information that the physicians WORKING at the VA Medical facilities SHOULD have gotten to evaluate my problem but refused and resorted to intimidation tactics which obvious DID NOT AND WILL NOT WORK!

Included you will find pictures that CLEARLY show trichorrhexis nosoda IS what is happening with my hair. In addition, I also submitted a case report that details HOW you begin to workup a patient for this problem and what you will notice is that she asked "if the body hair was involved"? My body hair is involved WHICH SHOULD HAVE LED TO KNOW THAT IT WAS NOT ACQUIRED BY PRODUCT OR RELAXER! In addition, the hair in the product case looks different microscopically and I was careful to print IN COLOR to highlight this FACT.

Next, I included lab testing that covered the basic WHICH INCLUDED PROTIEN. I was shown NOT to be protein deficient and she WAS WRONG TO suggest and increase without checking the urea cycle! Urea Cycle defect can be adult onset AND protein increases are detrimental to those having them. So to check for amino acid related issues I had an amino acids analysis done via another CLIA laboratory and my citrulline values are double the reference range and the thing that has not push this problem over the edge is that my ammonia has been miraculously kept in check over all but it is still not know if I could be experiencing periodic spikes causing the break to occur. My urine ammonia is relative low but my plasma ammonia is middling in value and according to an authority difficult to correctly obtain. I was sent instructions on how to have it performed correctly and will repeat as funds permit. Last, I had a CLIA laboratory perform a FULL VITAMIN assessment and NO MY BIOTIN IS NOT DEFICIENT EITHER.

With that in addition, to my award for ACTUALLY BEING CORRECT, I want the full names and license numbers of all physicians (including the outside one) so that I can report them to the NATIONAL MEDICAL ASSOCIATION as well as the NATIONAL DERMATOLOGICAL ASSOCIATION. What they've done was and continues to be wrong and it not only endangers my life but the many millions of other veterans that they get PAID TO PROPERLY EVALUATE AND TREAT!

Exhibit C

Department of
Veterans Affairs
Office of Regional Counsel
3322 West End Avenue Suite 509
Nashville TN 37203
OFFICIAL BUSINESS

PLACE STICKER AT TOP OF ENVELOPE TO THE RIGHT OF THE RETURN ADDRESS. FOLD AT DOTTED LINE

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KENDRIA Y. WEST
1109 BLUEWILLOW COURT
ANTIOCH, TN 37013

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Department of Veterans Affairs
Office of Chief Counsel
Southeast District – North
3322 West End Avenue, Suite 509
Nashville, TN 37203
Phone 615-695-4633
Fax 615-695-4634

December 3, 2015

Kendria Y. West
1109 Bluewillow Court
Antioch, TN 37013

CERTIFIED MAIL

Re: Administrative Tort Claim of Kendria Y. West
Our Case No. 11476

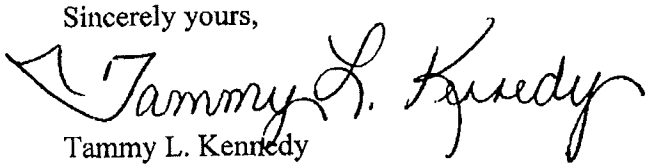
Dear Ms. West:

This office has completed an investigation of the above-captioned claim under the Federal Tort Claims Act (FTCA), and it is denied. The FTCA provides a legal remedy enabling an individual to recover damages under circumstances where the United States, if it were a private person, would be liable. Our investigation, which included a review by a dermatologist who was not involved in your care, did not find any negligent or wrongful act or omission on the part of a Department of Veterans Affairs (VA) employee acting within the scope of his or her employment that caused harm regarding the dermatology appointment on May 20, 2015 and the matters raised in your claim.

Further action on this matter may be instituted in accordance with the FTCA, sections 1346(b) and 2671-2680, title 28, United States Code (U.S.C.), which provide that a tort claim that is administratively denied may be presented to a federal district court for judicial consideration. Such a suit must be initiated within six months after the date of mailing of this notice of final denial as shown by the date of this letter (section 2401(b), title 28, U.S.C.). If such a suit is filed, the proper party defendant would be the United States, and not the VA.

Alternatively, a request for reconsideration of this claim by this office may be filed by: (1) mail to Office of General Counsel (021B), 810 Vermont Avenue, N.W., Washington, DC 20420; (2) fax to (202) 273-6385; or (3) e-mail to OGC.torts@mail.va.gov. VA must receive such a request within six months of the date of mailing of this notice of final denial as shown by the date of this letter. If a request for reconsideration is made, VA shall have six months from receipt of that request during which the option to file suit in an appropriate federal court under 28 U.S.C. 2675(a) is suspended.
Please note that FTCA claims are governed by a combination of federal and state laws. Some state laws may limit or bar a claim or law suit. VA legal staff handling FTCA claims work for the federal government, and cannot provide advice regarding the impact of state laws or state filing requirements.

Sincerely yours,



Tammy L. Kennedy
Chief Counsel

Exhibit C

CLAIM FOR DAMAGE, INJURY, OR DEATH

INSTRUCTIONS: Please read carefully the instructions on the reverse side and supply information requested on both sides of this form. Use additional sheet(s) if necessary. See reverse side for additional instructions.

FORM APPROVED
OMB NO. 1105-0008

1. Submit to Appropriate Federal Agency:

DEPARTMENT OF VETERAN AFFAIRS
OFFICE OF REGIONAL COUNSEL
3322 WEST END AVE, SUITE 509
NASHVILLE TN

2. Name, address of claimant, and claimant's personal representative if any. (See instructions on reverse). Number, Street, City, State and Zip code.

KENDRIA Y WEST
1109 BLUEWILLOW CT
ANTIOCH TN 37013

3. TYPE OF EMPLOYMENT	4. DATE OF BIRTH	5. MARITAL STATUS	6. DATE AND DAY OF ACCIDENT	7. TIME (A.M. OR P.M.)
[X] MILITARY [ ] CIVILIAN	10/05/1967	SINGLE	05/20/2015 WED	2:00 P.M.

8. BASIS OF CLAIM (State in detail the known facts and circumstances attending the damage, injury, or death, identifying persons and property involved, the place of occurrence and the cause thereof. Use additional pages if necessary).

SEE ATTACHED

9. **PROPERTY DAMAGE**

NAME AND ADDRESS OF OWNER, IF OTHER THAN CLAIMANT (Number, Street, City, State, and Zip Code).

N/A

BRIEFLY DESCRIBE THE PROPERTY, NATURE AND EXTENT OF THE DAMAGE AND THE LOCATION OF WHERE THE PROPERTY MAY BE INSPECTED. (See instructions on reverse side).

N/A

10. **PERSONAL INJURY/WRONGFUL DEATH**

STATE THE NATURE AND EXTENT OF EACH INJURY OR CAUSE OF DEATH, WHICH FORMS THE BASIS OF THE CLAIM. IF OTHER THAN CLAIMANT, STATE THE NAME OF THE INJURED PERSON OR DECEDENT.

Continued perforation of hair that IS NOT associated with relaxing as all of my hair does it. Continued emotional and psychological anguish from my hair breakage. Continued deterioration of my physical health from the underlying deficiency not being properly identified or addressed!

11. **WITNESSES**

NAME	ADDRESS (Number, Street, City, State, and Zip Code)
JEANETTE WEST	1109 BLUEWILLOW CT ANTIOCH TN 37013

12. (See instructions on reverse). **AMOUNT OF CLAIM** (in dollars)

12a. PROPERTY DAMAGE	12b. PERSONAL INJURY	12c. WRONGFUL DEATH	12d. TOTAL (Failure to specify may cause forfeiture of your rights).
0.00	999000000000	0.00	999000000000

I CERTIFY THAT THE AMOUNT OF CLAIM COVERS ONLY DAMAGES AND INJURIES CAUSED BY THE INCIDENT ABOVE AND AGREE TO ACCEPT SAID AMOUNT IN FULL SATISFACTION AND FINAL SETTLEMENT OF THIS CLAIM.

13a. SIGNATURE OF CLAIMANT (See instructions on reverse side).	13b. PHONE NUMBER OF PERSON SIGNING FORM	14. DATE OF SIGNATURE
	615-641-1919	02/08/2016

CIVIL PENALTY FOR PRESENTING FRAUDULENT CLAIM	CRIMINAL PENALTY FOR PRESENTING FRAUDULENT CLAIM OR MAKING FALSE STATEMENTS
The claimant is liable to the United States Government for a civil penalty of not less than $5,000 and not more than $10,000, plus 3 times the amount of damages sustained by the Government. (See 31 U.S.C. 3729).	Fine, imprisonment, or both. (See 18 U.S.C. 287, 1001.)

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Previous Edition is not Usable

NSN 7540-00-634-4046

STANDARD FORM 95 (REV. 2/2007)
PRESCRIBED BY DEPT. OF JUSTICE
28 CFR 14.2

95-109

Exhibit C

INSURANCE COVERAGE

In order that subrogation claims may be adjudicated, it is essential that the claimant provide the following information regarding the insurance coverage of the vehicle or property.

15. Do you carry accident Insurance? ☐ Yes If yes, give name and address of insurance company (Number, Street, City, State, and Zip Code) and policy number. ☒ No

N/A

16. Have you filed a claim with your insurance carrier in this instance, and if so, is it full coverage or deductible? ☐ Yes ☒ No

N/A

17. If deductible, state amount.

0.00

18. If a claim has been filed with your carrier, what action has your insurer taken or proposed to take with reference to your claim? (It is necessary that you ascertain these facts).

N/A

19. Do you carry public liability and property damage insurance? ☐ Yes If yes, give name and address of insurance carrier (Number, Street, City, State, and Zip Code). ☒ No

N/A

INSTRUCTIONS

Claims presented under the Federal Tort Claims Act should be submitted directly to the "appropriate Federal agency" whose employee(s) was involved in the incident. If the incident involves more than one claimant, each claimant should submit a separate claim form.

Complete all items - Insert the word NONE where applicable.

A CLAIM SHALL BE DEEMED TO HAVE BEEN PRESENTED WHEN A FEDERAL AGENCY RECEIVES FROM A CLAIMANT, HIS DULY AUTHORIZED AGENT, OR LEGAL REPRESENTATIVE, AN EXECUTED STANDARD FORM 95 OR OTHER WRITTEN NOTIFICATION OF AN INCIDENT, ACCOMPANIED BY A CLAIM FOR MONEY DAMAGES IN A **SUM CERTAIN** FOR INJURY TO OR LOSS OF PROPERTY, PERSONAL INJURY, OR DEATH ALLEGED TO HAVE OCCURRED BY REASON OF THE INCIDENT. THE CLAIM MUST BE PRESENTED TO THE APPROPRIATE FEDERAL AGENCY WITHIN **TWO YEARS** AFTER THE CLAIM ACCRUES.

Failure to completely execute this form or to supply the requested material within two years from the date the claim accrued may render your claim invalid. A claim is deemed presented when it is received by the appropriate agency, not when it is mailed.

If instruction is needed in completing this form, the agency listed in item #1 on the reverse side may be contacted. Complete regulations pertaining to claims asserted under the Federal Tort Claims Act can be found in Title 28, Code of Federal Regulations, Part 14. Many agencies have published supplementing regulations. If more than one agency is involved, please state each agency.

The claim may be filled by a duly authorized agent or other legal representative, provided evidence satisfactory to the Government is submitted with the claim establishing express authority to act for the claimant. A claim presented by an agent or legal representative must be presented in the name of the claimant. If the claim is signed by the agent or legal representative, it must show the title or legal capacity of the person signing and be accompanied by evidence of his/her authority to present a claim on behalf of the claimant as agent, executor, administrator, parent, guardian or other representative.

If claimant intends to file for both personal injury and property damage, the amount for each must be shown in item number 12 of this form.

The amount claimed should be substantiated by competent evidence as follows:

(a) In support of the claim for personal injury or death, the claimant should submit a written report by the attending physician, showing the nature and extent of the injury, the nature and extent of treatment, the degree of permanent disability, if any, the prognosis, and the period of hospitalization, or incapacitation, attaching itemized bills for medical, hospital, or burial expenses actually incurred.

(b) In support of claims for damage to property, which has been or can be economically repaired, the claimant should submit at least two itemized signed statements or estimates by reliable, disinterested concerns, or, if payment has been made, the itemized signed receipts evidencing payment.

(c) In support of claims for damage to property which is not economically repairable, or if the property is lost or destroyed, the claimant should submit statements as to the original cost of the property, the date of purchase, and the value of the property, both before and after the accident. Such statements should be by disinterested competent persons, preferably reputable dealers or officials familiar with the type of property damaged, or by two or more competitive bidders, and should be certified as being just and correct.

(d) **Failure to specify a sum certain will render your claim invalid and may result in forfeiture of your rights.**

PRIVACY ACT NOTICE

This Notice is provided in accordance with the Privacy Act, 5 U.S.C. 552a(e)(3), and concerns the information requested in the letter to which this Notice is attached.

A. *Authority:* The requested information is solicited pursuant to one or more of the following: 5 U.S.C. 301, 28 U.S.C. 501 et seq., 28 U.S.C. 2671 et seq., 28 C.F.R. Part 14.

B. *Principal Purpose:* The information requested is to be used in evaluating claims.

C. *Routine Use:* See the Notices of Systems of Records for the agency to whom you are submitting this form for this information.

D. *Effect of Failure to Respond:* Disclosure is voluntary. However, failure to supply the requested information or to execute the form may render your claim "invalid."

PAPERWORK REDUCTION ACT NOTICE

This notice is solely for the purpose of the Paperwork Reduction Act, 44 U.S.C. 3501. Public reporting burden for this collection of information is estimated to average 6 hours per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden, to the Director, Torts Branch, Attention: Paperwork Reduction Staff, Civil Division, U.S. Department of Justice, Washington, DC 20530 or to the Office of Management and Budget. Do not mail completed form(s) to these addresses.

Exhibit C

KENDRIA Y WEST
SF 95
BASIS OF CLAIM

On 05/20/15 I had an appointment to see the Dermatologist at the Alvin C York VA Medical Facility. She (Carmen Arzubiaga) didn't tell me her name, refused to look at my hair under the microscope and put her finger over the injured area whilst pretending to look at it under the big magnifying glass! The magnifying glass DOES NOT zero in like the microscope. Because of this, she failed to properly diagnose my hair shaft defect. My hair is perforating in spots so it's not a split ends issue! Perforations MUST BE a hair shaft defect but there are several that could be indicated. There is Bubble Hair, Beaded Hair/Monliethrix, Pili torti, Trichorrhexis invaginata, Trichorrhexis nodosa, or Trichothiodystrophy! She needed to use to microscope to get this correct and she didn't!

She told me to "try" some biotin and that she'd put in for it but didn't know if it would be approved. In addition, she told me that Biotin was NOT listed as a B vitamin! It IS listed as B7! I have attached the instructions that I was given at this appointment. This is usually typed into the computer and printed out but it wasn't this time. She also stated that I should "use lotion" instead of using harsh conditioners. Her advice is NOT sound considering that I use a natural baby shampoo and conditioner, don't relax my body hair but have the same problem there as well. In addition, high protein diets are taxing on the kidneys and she performed not one test that shows that I'm protein or biotin deficient! Just so that you understand that I understand a Biotin Deficiency workup consists of **biotin levels**, **serum ammonia levels**, **urine ketones levels**, **quantitative plasma amino acid levels**, **plasma carnitine levels**; **both free and total (sometimes termed free and esterified)** , and a **routine serum chemistry panel**! As such, it's NEVER really the doctor that determines what's wrong it's the TESTS which she failed to run!

Doctors are to properly evaluate patients to arrive at a proper and accurate DIAGNOSIS. Doctors are IN FACT scientists that use science to determine health problems. It is NOT APPROPRIATE and DANGEROUS to patient health to have patients to "try this and that" without properly identifying the problem. It is tantamount to malpractice because my problem still exists and causes not only psychological and emotional trauma but the further deterioration of the actual health deficiency! If you pay attention to the fact that she suggested a biotin deficiency and that work up includes serum ammonia level and quantitative plasma amino acid levels and you know anything you'd recognize that hair breakage like mine is characteristic of a urea cycle defect (which is curable but deadly) and the protocol testing which she failed to perform is seeking to find the actual deficit so that it can be corrected. Urea cycle defects are typically seen in children (I worked in pediatrics and I've seen a case of a 2 year old with one which is why I sought help) HOWEVER, it can occur in adults where it is termed "late onset" and triggered by illness! How long a patient will live is a matter of opinion, whether or not a patient is deficient in this, that or the other is a

Exhibit C

KENDRIA Y WEST
SF 95
BASIS OF CLAIM

MATTER OF FACT! So this is a FACTUAL case based on failure to perform her duty!

In addition, I demonstrate financial need but her request for the VA to supply my biotin was DENIED because she failed to demonstrate the need by performing appropriate and necessary tests! She only offered a cortisol test to see if it was the cause of my hyperpigmentation which is still wrong for that as well. Cortisol is best as saliva but can be serum or urine. In addition, the whole endocrine system would need to be evaluated; also cortisol fluctuates so there is am/pm and 24 hour. So she did nothing correct during my visit.

Based on this experience Dr Arzubiaga has failed me miserably and put my emotional, psychological AND physical well being into continued jeopardy! It is this FACT that is the basis for this form submission.

After I initially submitted a SF-95 regarding this I was re-seen (as I was to allow in an attempt to resolve this issue) on 09/17/15 by the Dermatology Clinic at the Nashville Facility. During this visit I saw a Dr. Livingood who proceeded to tell me that though she'd done NOTHING that "Dr Arzubiag had done a good job." and that "since I didn't have a job I wouldn't be able to get testing done to show that she didn't." He went on to go get Dr. Stricklin who told me FLAT OUT that he was going to "say the same thing because he didn't care if I re-filed because he had his 30 years and was retiring." So though went to the appointment and allowed them to correct the problem they chose to try to intimidate me and DID NOTHING!

In light of that situation I prepared to re-file and prepared to show THE INTENTIONAL AND MALICIOUS FAILURE of the Dermatology Department of BOTH FACILITIES in the Nashville Area. This continued failure made it essential for me to re-file, increase the requested damages, and SHOW that they were wrong!

I did receive notice from Tammy Kennedy that she was denying the initial request which was fine with me because of the progression of the problem. However, in her letter she stated the "an outside physician said that nothing wrong was done." This COULD HARDLY BE TRUE because it is NEVER not wrong to properly evaluate your patient. In addition, I NEVER SIGNED ANY RELEASE OF INFORMATION for ANY OTHER DOCTOR to review my information, so in doing so she violated my privacy rights. Also, trichorrhexis nodosa is a visual diagnosis and he/she most certainly didn't see me or my hair so it would be impossible for him/her to know if she was wrong or not!

So with those facts now documented, I also submit CLIA certified laboratory results and ACTUAL PICTURES OF MY HAIR PROBLEM so that as this case

proceeds the judge and jury SEE that I'm NOT participating in ANY case of he said, she said but am dealing with objective information that the physicians WORKING at the VA Medical facilities SHOULD have gotten to evaluate my problem but refused and resorted to intimidation tactics which obvious DID NOT AND WILL NOT WORK!

Included you will find pictures that CLEARLY show trichorrhexis nosoda IS what is happening with my hair. In addition, I also submitted a case report that details HOW you begin to workup a patient for this problem and what you will notice is that she asked "if the body hair was involved"? My body hair is involved WHICH SHOULD HAVE LED TO THE CONCLUSION THAT IT WAS NOT ACQUIRED BY PRODUCT OR RELAXER! In addition, the hair in the product case looks different microscopically and I was careful to print IN COLOR to highlight this FACT. The break is pulled and long in the product case and doesn't paint brush out like in the medical case like mine does.

Next, I included lab testing that covered the basic WHICH INCLUDED PROTIEN. I was shown NOT to be protein deficient and she WAS WRONG TO suggest and increase without checking the urea cycle! Urea Cycle defect can be adult onset AND protein increases are detrimental to those having them. So to check for amino acid related issues I had an amino acids analysis done via another CLIA laboratory and my citrulline values are double the reference range and the thing that has not push this problem over the edge is that my ammonia has been miraculously kept in check over all but it is still not know if I could be experiencing periodic spikes causing the break to occur. My urine ammonia is relative low but my plasma ammonia is middling in value and according to an authority difficult to correctly obtain. I was sent instructions on how to have it performed correctly and will repeat as funds permit. Last, I had a CLIA laboratory perform a FULL VITAMIN assessment and NO MY BIOTIN IS NOT DEFICIENT EITHER.

With THESE FACTS and my award for ACTUALLY BEING CORRECT, I want the full names and license numbers of all physicians (including the outside one) so that I can report them to the NATIONAL MEDICAL ASSOCIATION as well as the NATIONAL DERMATOLOGICAL ASSOCIATION. What they've done was and continues to be wrong and it not only endangers my life but the lives of the many millions of other veterans that they get PAID TO PROPERLY EVALUATE AND TREAT! Patients go to the doctor to be evaluated NOT to have the doctor GUESS AND NOT TEST!

Exhibit C

VA
U.S. Department of Veterans Affairs
810 Vermont Ave. NW
Washington, DC 20420

Washington DC 20420
OFFICIAL BUSINESS

7016 1370 0002 2209 3562

CERTIFIED MAIL

Ms. Kendria Y. West
1109 Bluewilliow Court
Antioch, TN 37013

Exhibit C

CAP DISTRICT
NO 207
25 MAY 17
PM 3 1

15 77

Exhibit C

VA | U.S. Department of Veterans Affairs

Office of the General Counsel
Washington DC 20420

MAY 25 2017

In Reply Refer To: 021B: GCL: #11476

Certified Mail

Ms. Kendria Y. West
1109 Bluewillow Court
Antioch, TN 37013

Re: Administrative Tort Claim- Request for Reconsideration

Dear Ms. West:

This office has completed reconsideration of the above-referenced matter under the Federal Tort Claims Act (FTCA), and it is again denied.

The FTCA provides a legal remedy enabling an individual to recover damages under circumstances where the United States, if it were a private person, would be liable. Our review, which included consideration of the opinion of a dermatology specialist, who was not involved in your care, revealed no negligent or wrongful act or omission, by any Department of Veterans Affairs (VA) employee with respect to diagnosis or treatment of your hair condition, on or about May 20, 2015 and/or September 17, 2015.

Further action on the matter may be instituted in accordance with the FTCA, sections 1346(b) and 2671-2680, title 28, United States Code, which provides, in effect, that a tort claim that is administratively denied may be presented to a Federal district court for judicial consideration. Such a suit must be initiated, however, within 6 months after the date of mailing of this notice of final denial as shown by the date of this letter (section 2401(b), title 28, United States Code). If such a suit is filed, the proper party defendant would be the United States, not VA.

Please note that FTCA claims are governed by a combination of Federal and state laws. Some state laws may limit or bar a claim or law suit. VA attorneys handling FTCA claims work for the Federal government, and cannot provide advice regarding the impact of state laws or state filing requirements.

Sincerely yours,

Pamela Anne-Allen

for
E. Douglas Bradshaw, Jr.
Chief Counsel, Torts Law Group

Physician SOAP Notes

SOAP Note Programs
eMedNotes - Automated Physcian Progress Notes

What Does SOAP Stand For?

1. **SUBJECTIVE** — The initial portion of the SOAP note format consists of subjective observations. These are symptoms the patient verbally expresses or as stated by a significant other. These subjective observations include the patient's descriptions of pain or discomfort, the presence of nausea or dizziness, when the problem first started, and a multitude of other descriptions of dysfunction, discomfort, or illness the patient describes.

2. **OBJECTIVE** — The next part of the format is the objective observation. These objective observations include symptoms that can actually be measured, seen, heard, touched, felt, or smelled. Included in objective observations are vital signs such as temperature, pulse, respiration, skin color, swelling and the results of diagnostic tests.

3. **ASSESSMENT** — Assessment follows the objective observations. Assessment is the diagnosis of the patient's condition. In some cases the diagnosis may be clear, such as a contusion. However, an assessment may not be clear and could include several diagnosis possibilities.

4. **PLAN** — The last part of the SOAP note is the health care provider's plan. The plan may include laboratory and/or radiological tests ordered for the patient, medications ordered, treatments performed (e.g., minor surgery procedure), patient referrals (sending patient to a specialist), patient disposition (e.g., home care, bed rest, short-term, long-term disability, days excused from work, admission to hospital), patient directions (e.g. elevate foot, RTO 1 week), and follow-up directions for the patient.

What IS a SOAP Note?

The SOAP note format is used to standardize medical evaluation entries made in clinical records. The SOAP note is written to facilitate improved communication among all involved in caring for the patient and to display the assessment, problems and plans in an organized format. Many Electronic Health Records (EHR) systems are capable of producing SOAP Notes. The actual notes and other information contained within the EMR are commonly referred to as Electronic Medical Records or EMRs. Here's more information on EHRs

Exhibit D

Components of a SOAP Note?

Other Examples
eMedNotes Psychiatric Note
DocStoc.com Example
Univ of Kansas School of Nursing

The four components of a SOAP note are **S**ubjective, **O**bjective, **A**ssessment, and **P**lan. The length and focus of each component of a SOAP note varies depending on the specialty; for instance, a surgical SOAP note will generally be much briefer than a psychiatric SOAP note, and will focus on issues that relate to post-surgical status.

Subjective component

This describes the patient's current condition in narrative form. The history or state of experienced symptoms are recorded in the **patient's own words**.
It will include all pertinent and negative symptoms under review of body systems in addition pertinent medical history, surgical history, family history, social history along with current medications and allergies are also recorded.
A SAMPLE history is one method of obtaining this information from a patient.
If this is the first time a doctor is seeing a patient, they will take a **H**istory of **P**resent **I**llness or HPI.
To structure this portion of the note, you can use another mnemonic: OLD CHARTS, as in what would you find if you looked at the patient's "old chart"

- **O**nset
- **L**ocation
- **D**uration
- **CH**aracter (sharp, dull, etc)
- **A**lleviating/Aggravating factors
- **R**adiation
- **T**emporal pattern (every morning, all day, etc)
- **S**ymptoms associated

Objective component

The objective component includes:

- Vital signs
- Findings from physical examinations, such as posture, bruising, and abnormalities
- Results from laboratory tests
- Measurements, such as age and weight of the patient.

Assessment

Is a quick summary of the patient with main symptoms/diagnosis including a differential diagnosis, a

list of other possible diagnoses usually in order of most likely to least likely. When used in a **Problem Oriented Medical Record**, relevant problem numbers or headings are included as subheadings in the assessment.

What is a Problem Oriented Medical Record

A **P**roblem **O**riented **M**edical **R**ecord (**POMR**), a method of recording data about the health status of a patient in a problem-solving system. The POMR preserves the data in an easily accessible way that encourages ongoing assessment and revision of the health care plan by all members of the health care team.

The particular format of the system used varies from setting to setting, but the components of the method are similar. A data base is collected before beginning the process of identifying the patient's problems. The data base consists of all information available that contributes to this end, such as that collected in an interview with the patient and family or others, that from a health assessment or physical examination of the patient, and that from various laboratory and radiologic tests.

It is recommended that the data base be as complete as possible, limited only by potential hazard, pain or discomfort to the patient, or excessive assumed expense of the diagnostic procedure. The interview, augmented by prior records, provides the patient's history, including the reason for contact; an identifying statement that is a descriptive profile of the person; a family illness history; a history of the current illness; a history of past illness; an account of the patient's current health practices; and a review of systems.

The physical examination or health assessment makes up the second major part of the data base. The extent and depth of the examination vary from setting to setting and depend on the services offered and the condition of the patient.

The next section of the POMR is the **master problem list**.

The formulation of the problems on the list is similar to the assessment phase of the nursing process. Each problem as identified represents a conclusion or a decision resulting from examination, investigation, and analysis of the data base. A problem is defined as anything that causes concern to the patient or to the caregiver, including physical abnormalities, psychologic disturbance, and socioeconomic problems. The master problem list usually includes active, inactive, temporary, and potential problems. The list serves as an index to the rest of the record and is arranged in five columns: a chronologic list of problems, the date of each problem's onset, the action taken, the outcome (often its resolution), and the date of the outcome. Problems may be added, and intervention or plans for intervention may be changed; thus the status of each problem is available for the information of all members of the various professions involved in caring for the patient.

The third major section of the POMR is the initial plan, in which each separate problem is named and described, usually on the progress note in a SOAP format: S, subjective data from the patient's point of view; O, the objective data acquired by inspection, percussion, auscultation, and palpation and from laboratory and radiologic tests; A, assessment of the problem that is an analysis of the subjective and objective data; and P, the plan, including further diagnostic work, therapy, and education or counseling. After an initial plan for each problem is formulated and recorded, the problems are followed in the progress notes by narrative notes in the SOAP format or by flow sheets showing the significant data in a tabular manner.

A discharge summary is formulated and written, relating the overall assessment of progress during treatment and the plans for follow-up or referral. The summary allows a review of all the problems initially identified and encourages continuity of care for the patient.

Plan

This is what the health care provider will do to treat the patient's concerns. This should address each item of the differential diagnosis. A note of what was discussed or advised with the patient as well as timings for further review or follow-up may also be included. Often the Assessment and Plan sections are grouped together.

SOAP notes facilitate better medical care when used in the patient's record and provide for far greater review and quality control. **SOAP Note** Documentation of patient complaints and treatment should be consistent, concise and comprehensive.

Conclusion

The **SOAP note** is not meant to be as detailed as a Progress Report. Partial sentences and abbreviations are appropriate. However, care should be exercised based on how the abbreviations are used as they can differ for each specialty. The length of the note will differ for each specialty as well.

SOAP notes can be flexible and different care providers will often have their own styles as well as different office will have thier preferences. Usually **SOAP Notes** written by the uninitated will usually be a little longer than those of more advanced staff with more clinical judgment and experience in proper SOAP note writing format. A short, precise SOAP note is often better than an entry that is too verbose.

Documenting patient encounters in the medical record is an integral part of practice workflow. Additionally, Prehospital care providers such as EMTs may use the same or similar format to communicate patient information to Emergency department personell.

Examples

Very rough example for a patient being reviewed following an appendectomy (resembles a surgical SOAP note).

Surgery Service, Dr. Jones

S: No Chest Pain or Shortness of Breath. "Feeling better today." Patient reports flatus.

O: Afebrile, P 84, R 16, BP 130/82. No acute distress. Neck no JVD, Lungs clear Cor RRR Abd Bowel sounds present, mild RLQ tenderness, less than yesterday. Wounds look clean. Ext without edema

A: Patient is a 37 year old man on post-operative day 2 for laparoscopic appendectomy, recently passed flatus.

P: Recovering well. Advance diet. Continue to monitor labs. Prepare for discharge home tomorrow morning.

Note that the plan itself includes various components:
Diagnostic component - continue to monitor labs
Therapeutic component - advance diet
Patient education component - that is progressing well
Disposition component - discharge to home in the morning

Progress Notes

IMMUNIZATIONS: (VET DECLINE INFLUENZA VACCINE).

HEPA AD	12/01/2005	MURFREESBO*
	06/03/2005	MURFREESBO*
TD-ADULT	01/17/2012	MURFREESBO*
	00/00/2000	@ private *

5. VITAMIN D LEVEL ORDERED AND SHE WILL GET FROM LAB.

6. ALLERGY CONCERNS- VET STATES SHE CAN FEEL HISTAMINE RELEASE-(SHE HAS A HARD TIME DESCRIBING -RUNNY NOSE/ BODY RACING FEEL / CONGESTION). SHE DESCRIBES NOT DOING WELL WITH MEDICATIONS (DISUSSED CLARITIN/ZYRTEC/ALLEGRA ETC..)SHE WOULD LIKE TO SEE ALLERGIST. VET REQUEST.

7. LABS ARE NORMAL. PLEASE GIVE VET A COPY.

8.Hyperpigmentation and trichorrhexia.Patient broght lots of literature from WEB MD ans wants tests for urea cycle disorder,parasite infestation in GI(Liver fluke) and mitochondrial disease etc.Reassured her in abscence of significant labs but not convinced of my concern of huge differential diagnostic possibilities to NIL.Will ask dermatology to check her complaint of trichorrhexia and will check her stool for ova parasite.

/es/ ASHISH K CHAKRAVARTHY MD
ATTENDING
Signed: 02/09/2015 14:00

LOCAL TITLE: CLINICAL REMINDER FOLLOW-UP
STANDARD TITLE: NURSING NOTE
DATE OF NOTE: FEB 09, 2015@13:10 ENTRY DATE: FEB 09, 2015@13:10:22
AUTHOR: BURKHART,DARRELL E EXP COSIGNER:
URGENCY: STATUS: COMPLETED

Homelessness Screening:
In the past 2 months, have you been living in stable housing that you own, rent, or stay in as part of a household? Yes - Living in stable housing.
Are you worried or concerned that in the next 2 months you may NOT have stable housing that you own, rent, or stay in as part of a household?
No - Not worried about housing near future
Alcohol Screening:
Alcohol Screening
How often did you have a drink containing alcohol in the past year?

Progress Notes

The patient, family, and/or caregiver has been educated on new medications including common and severe adverse reactions and/or side effects by the prescribing provider. Yes
Release of Information:

/es/ DARRELL (GENE)BURKHART,LPN
PRIMARY CARE
Signed: 02/09/2015 14:08

LOCAL TITLE: PRIMARY CARE - FOLLOW-UP
STANDARD TITLE: PRIMARY CARE NOTE
DATE OF NOTE: FEB 09, 2015@13:30 ENTRY DATE: FEB 09, 2015@13:31:01
AUTHOR: CHAKRAVARTHY,ASHISH EXP COSIGNER:
URGENCY: STATUS: COMPLETED

CC :hyperpigmentation of skin both dorsum hands and knuckle and also around left lower lip Xyrs

HPI:47 BF presented to my office for her outine annual check up.She has noticed some darkening of skin color as described with no other associated symptome.She has regular period.No children and never been pregnent.Denies any expoaures or use of chemicals.No arthralgia myalgias.No fatigues rash or skin lesions anywhere.No cough cold fever chills.No otc meds only medication is synthroid.Multiple other routine supplemental vitamins regularly.

ROS :All Other systems review : neg

PMH:

Abdominal Pain, Epigastric (ICD-9-CM 789Undifferentiated somatoform disorder (ICD-9-CM 300.82)
Neck Pain (ICD-9-CM 723.1) Pain in joint involving shoulder region (ICD-9-CM 719.41)
Osteoarthrosis involving the knee (ICD-9Dyspnea (ICD-9-CM 786.05)
Other Malaise and Fatigue (ICD-9-CM 780.Palpitations (ICD-9-CM 785.1)
Fibromyomata Uteri (ICD-9-CM 218.9) Mitral Valve Prolapse (ICD-9-CM 424.0)
Tachycardia (ICD-9-CM 785.0) Hypercholesterolemia (ICD-9-CM 272.0)
Hypothyroidism (ICD-9-CM 244.9)

SH:Single with no children.No risk for HIV or hepatitis .
Smoking()no
ETOH()no
Drugs()
no
FH:

MEDICAL RECORD — Progress Notes

NOTE DATED: 01/17/2012 13:54
LOCAL TITLE: MEDICATION RECONCILIATION
STANDARD TITLE: MEDICATION MGT NOTE
VISIT: 01/17/2012 15:30 MU-PCC/MEADOWS/RIN/FIRM C
Medication Review:
Active Outpatient Medications (including Supplies):

	Pending Outpatient Medications	Status
1)	SELENIUM SULFIDE 2.5% LOTION/SHAMPOO APPLY 1 CAPFUL TO SHAMPOO SCALP AS DIRECTED - FOR DANDRUFF/DERMATITIS	PENDING

All current/active medications have been reviewed with the patient and are correct as listed Yes

The Patient has been provided a list of current/active medication
Yes

Release of Information:
Patient declined the option of having his/her active medication list sent to outside provider(s).

DRAFT COPY - DRAFT COPY -- ABOVE NOTE IS UNSIGNED-- DRAFT COPY - DRAFT COPY

WEST,KENDRIA Y [redacted] — AC York, Murfreesboro, TN — Pt Loc: OUTPATIENT — Printed:01/17/2012 13:54 Vice SF 509

MEDICAL RECORD — Progress Notes

NOTE DATED: 01/17/2012 13:18
LOCAL TITLE: PRIMARY CARE - FOLLOW-UP
STANDARD TITLE: PRIMARY CARE NOTE
VISIT: 01/17/2012 15:30 MU-PCC/MEADOWS/RIN/FIRM C

42 YO HERE FOR F/U VISIT. SHE HAS HYPOTHYRODISM.

1. HYPOTHYRODISM- TSH IS NORMAL AND FREE T4 NORMAL TODAY. RT3 AND THYROGLOBULIN AB PENDING. SHE IS CURRENTLY TAKING 1/4 OF A TABLET OF LEVOTHYROXINE. HER LEVELS ARE NORMAL.

2. MVP WITH PALPITATIONS- CON'T ATENOLOL.

3. WILL SEND VET FOR MMG.~ Mammogram

4. F/U IN 5 MONTHS WITH FASTING LABS.

5. SEBORRHEA- SELENIUM PRESCRIBED.

6. HM- SHE WILL GET PAP AND MMG DONE LOCALLY.

PROV Mammogram Screening:
Mammogram Screening - Bilateral ordered to be performed at Murfreesboro.
PROV Pap Smear Screening:
The patient declined a PAP smear.
Comment: SHE WILL GET DONE LOCALLY.
PROV Cholesterol Screen:
Cholesterol test ordered.

Signed by: /es/ NATASHA D MEADOWS
MD
01/17/2012 13:45

01/17/2012 13:45 ADDENDUM STATUS: COMPLETED
VET REQUEST LAB RESULTS BE MAILED.
Signed by: /es/ NATASHA D MEADOWS
MD
01/17/2012 13:45

Lab # 31590

WEST,KENDRIA Y [redacted] AC York, Murfreesboro, TN Printed:01/17/2012 13:54
Pt Loc: OUTPATIENT Vice SF 509

Exhibit G

CSC

SPl

558-102

(See Instructions on Back of this Sheet)

NSN 7540-00-[illegible]

EMERGENCY CARE AND TREATMENT
(Medical Record)

TREATMENT FACILITY	U. S. N. H. OKINAWA JAPAN
LOG NUMBER	382110
ARRIVAL DATE (DAY / MONTH / YR.)	29 / 3 / 88
ARRIVAL TIME	1423
TRANSPORTATION TO HOSPITAL (Attach care enroute sheet)	☑ PRIVATE VEHICLE ☐ AMBULANCE ☑ OTHER (Specify)
CURRENT MEDS (Include immunization and other data)	NONE
HISTORY OBTAINED FROM	☑ PATIENT ☐ OTHER (Specify)
ALLERGIES	NKA
PATIENT'S HOME ADDRESS OR DUTY STATION (incl. State and ZIP Code)	#2861 USNH FPO SW 98778
HOME TELE. NO. (Inc. area code)	NONE
CHIEF COMPLAINT(S) (Include symptom(s), duration)	Stomach pain
SEX	F
AGE	21
POSSIBLE THIRD PARTY PAYER?	☐ YES ☑ NO
TIME SEEN BY PROVIDER	1510

VITAL SIGNS

TIME	1433
BP	102/99
PULSE	80
RESP.	18
TEMP.	98.8
WT. (Child)	

CATEGORY (See reverse)

- EMERGENT
- URGENT
- X NON-URGENT

ORDERS	INIT'S	TIME
GI cocktail	[illegible]	1515

ASSESSMENT/DIAGNOSIS

Recurrent indigestion

DESCRIBE (1) Subjective data (Pertinent History); (2) Objective data (Examination - include results of tests and x-rays); (3) Assessment (Diagnosis); (4) Plan (Treatment/Procedures - include medication given and follow-up)

21 y/o ♀ c/o hx of mid-epigastric pain of uncertain etiology, well treated ē previous command ž Mylanta, had neg UGI series 9 mos ago. No nausea, no diarrhea, no [illegible] or melena. Did questionable hyper-acidity at prev command

Exam: WD WN ♀, thin, in NAD
Lungs clr [illegible]
Abd NABS, soft, mid-epigastric pain only, no guarding or rebound.

1° p̄ GI cocktail - no probs.

Will refill Mylanta, may need to consider endoscopy if pain persists.

86 ND
ND ND

(CONTINUE ON SF 507, IF NEEDED)

DISPOSITION (Check all that apply)

- HOME
- ✓ FULL DUTY
- QUARTERS: 24 Hrs. / 48 Hrs. / 72 Hrs.
- MODIFIED DUTY UNTIL: DAY / MONTH / YEAR
- REFERRED TO (Indicate clinic)
- EMERGENCY / TODAY / 72 HOURS / ROUTINE
- ADMIT. TO HOSP. UNIT/SERVICE

CONDITION UPON RELEASE

- ✓ IMPROVED
- UNCHANGED
- DETERIORATED

TIME OF RELEASE: 1610

SIGNATURE OF PROVIDER AND ID STAMP: [signature] MD

PATIENT'S IDENTIFICATION (Mechanical imprint) FOR WRITTEN ENTRIES give: Name - last, first, middle; SSN; DOB; service status; grade and relation of sponsor or next of kin. (IMPORTANT: LIST FACILITY HOLDING TREATMENT RECORD).

WEST Kandi/ia [redacted]
[redacted]
Hm3 1N140 304
Pediatrics Lester
[illegible] FPO SEATTLE

INSTRUCTIONS TO PATIENT (Include medications ordered, any limitations and follow-up plans)

Mylanta 30 cc 4x/day as needed
Return to sick call if pain worsens

EMERGENCY CARE AND TREATMENT

STANDARD FORM 558 (REV. 6-82)
Prescribed by GSA and ICMR

RECORD BEFORE THE AGENCY (RBA)

00424

NSN 7540-00-634-4178 — 600-108

HEALTH RECORD — **CHRONOLOGICAL RECORD OF MEDICAL CARE**

DATE	SYMPTOMS, DIAGNOSIS, TREATMENT, TREATING ORGANIZATION (Sign each entry)
0740 29 JUNE 88	STAFF SICK CALL US NAVAL HOSPITAL OKINAWA, JAPAN FPO SEATTLE,WA 98778 T 97.7 P 76 R 16 B/P 94/64 Allergies NKDA Current meds none Complaint Epigastric pain

5/21 go @ USN, AD presents c % pain (burning sensation) to the epigastric region. Pt. seen on 29 March 1988 for same and was given a G.I. cocktail and 1° p ingestion related & further problems. Pt was given Mylanta @ that time and told to return to sick call if pain worsened. Further note by m.o. stating if pain persist may need to do endoscopy. Pt. denies N/V, diarrhea, pain radiation, worsening p meals or any positions causing aggravation of pain. Has been taking c antacids in past but now seem to be of little help. States pain duration is approx. 3'-5' and leaves for approx. 15' and then reoccurs. Period of episode usually last for 8° period each day. Denies any stress in her day to day activities. Relates everything is relatively normal. Noted on OTR dtd 29 March 88 that UGI performed @ last command x 9 months was negative. Continues to deny hematochezia or melena. Dx'd "Questionable

PATIENT'S IDENTIFICATION (Use this space for Mechanical Imprint)

RECORDS MAINTAINED AT:

PATIENT'S NAME (Last, First, Middle Initial)		SEX
WEST, KENDRIA		FEMALE
RELATIONSHIP TO SPONSOR	STATUS	RANK/GRADE
	AD	HM3
SPONSOR'S NAME		ORGANIZATION
		USNH
DEPART./SERVICE	[redacted]	DATE OF BIRTH
		[redacted]

CHRONOLOGICAL RECORD OF MEDICAL CARE — STANDARD FORM 600 (REV. 5-84) Prescribed by GSA and ICMR FIRMR (41 CFR) 201-45.505

RECORD BEFORE THE AGENCY (RBA)

00418

Exhibit G

NSN 7540-00-634-4176 — 600-108

HEALTH RECORD | **CHRONOLOGICAL RECORD OF MEDICAL CARE**

DATE	SYMPTOMS, DIAGNOSIS, TREATMENT, TREATING ORGANIZATION *(Sign each entry)*
0805	STAFF SICK CALL US NAVAL HOSPITAL — Allergies NKDA
5JULY88	OKINAWA, JAPAN FPO SEATTLE, WA 98778 — Current meds none
	T 97.8 P 68 R 16 B/P 90/60 — Complaint Epigastric Pain
	S/ Pt. returns for same complaint as 29 June 88. States she only took medication x1 per day and that she has had all the same meds before. Denies using any mylanta. Pt. seems very uncooperative c̄ Tx plan. States condition unchanged. States "I only eat one meal a day."
	O/ Alert, w/o x 3, afebrile, in Ø distress. Exam deffered. Pt. does not look at me when conversing only stares at the floor c̄ hands clasped in lap.
	A/ Pyrosis due to ? diet ? social problem.
	P/ Pt. reassured and importance of Tx explained. Spoke c̄ Dr. Mcddan and states Δ Tagamet to iii tabs P.O. QD @ H.S. x 6 wks. Reiterated that she will improve but takes time. Again stressed importance of taking meds. If Ø improvement will consider UGI + Internal Medicine Consult. RTC PRN or x 6 wk. MD A. McIntosh HMC
	MARK A. McINTOSH, HMC, [redacted] 5352

PATIENT'S IDENTIFICATION *(Use this space for Mechanical Imprint)*

Reviewed
Auerbach
A.J. AUERBACH MC
[redacted]8895

RECORDS MAINTAINED AT:

PATIENT'S NAME *(Last, First, Middle initial)*		SEX
WEST KENDRICK		FEMALE
RELATIONSHIP TO SPONSOR	STATUS	RANK/GRADE
SPONSOR'S NAME		ORGANIZATION USNH
DEPART./SERVICE Peds	SSN/IDENTIFICATION NO. [redacted]	DATE OF BIRTH

CHRONOLOGICAL RECORD OF MEDICAL CARE — STANDARD FORM 600 (REV. 6-84) Prescribed by GSA and ICMR FIRMR (41 CFR) 201-45.505

RECORD BEFORE THE AGENCY (RBA)

00416

Progress Notes

DATE OF NOTE: JUN 17, 2014@11:07:18 ENTRY DATE: JUN 17, 2014@11:07:18
AUTHOR: SIMMONS,JORY S SR EXP COSIGNER:
URGENCY: STATUS: COMPLETED

COMPENSATION AND PENSION EXAMINATION REPORT (FREE TEXT)

The RO examiner will be providing an addendum at the Nashville VA Regional Office, so the VAMC does not need to schedule an exam.
Refer questions to Chuck Weatherman at (615) 695-6014

Is the evidence of record sufficient to provide a diagnosis of celiac disease? (Rubber band around evidence.

Medical Opinion:

Based on evidence of record and gastrointestinal symptoms reported by veteran it is at least as likely as not that a diagnosis of Celiac Disease is appropriate. HLA-DQB1 Testing reveals veteran has one of the main genes that are associated with gluten sensitivity and Celiac Sprue disease.

/es/ JORY S SIMMONS SR,MD
C & P PHYSICIAN
Signed: 06/17/2014 11:07

LOCAL TITLE: C&P EXAMINATION
STANDARD TITLE: C & P EXAMINATION NOTE
DATE OF NOTE: JUN 04, 2014@15:25:08 ENTRY DATE: JUN 04, 2014@15:25:08
AUTHOR: ELLIS,SHELLEY EXP COSIGNER:
URGENCY: STATUS: COMPLETED

COMPENSATION AND PENSION EXAMINATION REPORT (FREE TEXT)

Are the veterans complaints of digestive problems the first manifestations of her now diagnosed celiac disease.

clarification:
I have reviewed cfile and recent exam. veteran has celiac disease. celiac disease is an autoimmune disease manifest as abdominal discomfort, chronic constipation and diarrhea, fatigue. these digestive problems began in service and persisted and were eventually diagnosed as celiac disease. thus, it is at least as likely as not that current celiac disease is related to GI problems in service.

Consult Requests

Printed On Oct 23, 2017

Current PC Provider: OKEKE-NNAMAH,UCHENNA S
Current PC Team: MU PCP 02 ACA
Current Pat. Status: Outpatient
Primary Eligibility: SC LESS THAN 50%(VERIFIED)
Patient Type: SC VETERAN
OEF/OIF: NO

[redacted]

Order Information
To Service: PADR NON FORMULARY/RESTRICTED MED ROUTINE
From Service: MU-DERMATOLOGY NEW
Requesting Provider: ARZUBIAGA,CARMEN M
Service is to be rendered on an OUTPATIENT basis
Place: Consultant's choice
Urgency: Within 72 hrs
Clinically Ind. Date: May 20, 2015
Orderable Item: PADR NON FORMULARY/RESTRICTED MED ROUTINE
Consult: Consult Request
Provisional Diagnosis: hair breakage
Reason For Request:
Have you already ENTERED the corresponding medication order?
Yes

Service: dermatology

Attending: C arzubiaga Md

Non-Formulary/Restricted drug/item requested:
(Please provide the following information for Pharmacy Service use should the requested drug be approved):

Drug Name: biotin 300mcg
Dosage: one po qd

If the consult is denied, it is your responsibility to contact the patient and notify them of the denial.

No formulary alternative exists.

Inter-facility Information
This is not an inter-facility consult request.

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at ALVIN C. YORK VAMC

Consult Requests

Printed On Oct 23, 2017

Status: COMPLETE
Last Action: COMPLETE/UPDATE

Facility Activity	Date/Time/Zone	Responsible Person	Entered By
CPRS RELEASED ORDER	05/20/15 15:12	ARZUBIAGA,CARMEN	ARZUBIAGA,CARMEN
COMPLETE/UPDATE	05/20/15 15:32	UNGAR,APRIL Q	UNGAR,APRIL Q
Note# 48074443			

Note: TIME ZONE is local if not indicated

LOCAL TITLE: PADR NON-FORMULARY MED CONSULT ROUTINE DENIED
STANDARD TITLE: PHARMACY CONSULT
DATE OF NOTE: MAY 20, 2015@15:30 ENTRY DATE: MAY 20, 2015@15:30:50
AUTHOR: UNGAR,APRIL Q EXP COSIGNER:
URGENCY: STATUS: COMPLETED

*** PADR NON-FORMULARY MED CONSULT ROUTINE DENIED Has ADDENDA ***

Denied

This is a dietary supplement and can be purchased OTC. VA does not provide meds or supplements for cosmetic reasons.

/es/ APRIL Q UNGAR
Pharm.D., BCPS, Clinical Pharmacist
Signed: 05/20/2015 15:32

Receipt Acknowledged By:
05/20/2015 15:39 /es/ CARMEN M ARZUBIAGA, MD
M.D

05/22/2015 ADDENDUM STATUS: COMPLETED

Contacted veteran and informed her the Biotin was not approved and per MD recommendation she should purchase over the counter. Veteran verbalized understanding.

Also, reminded veteran of need for am blood draw for cortisol level. Veteran states she will be seeing Endocrinology in July and plans to "let them take care of it".

/es/ LISA D EARP
RN

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at ALVIN C. YORK VAMC

Consult Requests

Signed: 05/22/2015 13:04

Receipt Acknowledged By:
05/22/2015 13:13 /es/ CARMEN M ARZUBIAGA, MD
M.D

==========

========== END ==========

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at ALVIN C. YORK VAMC

Progress Notes

LOCAL TITLE: PADR NON-FORMULARY MED CONSULT ROUTINE DENIED
STANDARD TITLE: PHARMACY CONSULT
DATE OF NOTE: MAY 20, 2015@15:30 ENTRY DATE: MAY 20, 2015@15:30:50
AUTHOR: UNGAR,APRIL Q EXP COSIGNER:
URGENCY: STATUS: COMPLETED

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/es/ APRIL Q UNGAR
Pharm.D., BCPS, Clinical Pharmacist
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05/20/2015 15:39 /es/ CARMEN M ARZUBIAGA, MD
M.D

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Also, reminded veteran of need for am blood draw for cortisol level. Veteran states she will be seeing Endocrinology in July and plans to "let them take care of it".

/es/ LISA D EARP
RN
Signed: 05/22/2015 13:04

Receipt Acknowledged By:
05/22/2015 13:13 /es/ CARMEN M ARZUBIAGA, MD
M.D

LOCAL TITLE: NURSING DERMATOLOGY CLINIC EXIT (BP)
STANDARD TITLE: DERMATOLOGY NURSING OUTPATIENT NOTE
DATE OF NOTE: MAY 20, 2015@14:47 ENTRY DATE: MAY 20, 2015@14:48:01
AUTHOR: EARP,LISA D EXP COSIGNER:
URGENCY: STATUS: COMPLETED

*** NURSING DERMATOLOGY CLINIC EXIT (BP) Has ADDENDA ***

Progress Notes

DISEASE PROCESS BEING TREATED:
[ ]ACTINIC KERATOSIS
[ ]R/O SKIN CANCER WITH BIOPSY
[X]OTHER
skin hyperpigmentation, scalp hair breaking

PROCEDURE DONE: No
[ ]SHAVE BIOPSY
[ ] PUNCH BIOPSY
[ ] CRYO
[ ] EXCISION
[ ] ED & C
NOTES:
PT AWARE THAT THEY CAN TAKE TYLENOL EVERY 4-6 HOURS IF THEY DEVELOP PAIN AFTER THE PROCEDURE OR CALL THE DERMATOLOGY CLINIC OR TELEPHONE CARE AFTER HOURS.

PAIN SCORE (POST-PROCEDURE ONLY) (1-10):

SUTURES TO BE REMOVED: Not applicable

[ ] KIT GIVEN
[ ] SUTURE REMOVAL INSTRUCTION GIVEN WITH DATE TO REMOVE

NEW MEDICATION REVIEWED: BY MD Non-formulary consult submitted for Bio
[X] NEW MEDS PRESCRIBED AND REVIEWED BY MD
[ ] NO NEW MEDS
[X] IF APPLICABLE, NEW MEDICATION RECONCILIATION SHEET PROVIDED FROM REPORTS TAB

WRITTEN WOUND CARE INSTRUCTIONS REVIEWED VERBALLY AND THEN GIVEN TO VETERAN:

Not applicable

PATIENT EDUCATION HANDOUTS REVIEWED AND GIVEN: Not applicable

[ ]Sun Protection Guidelines
[ ]Skin Cancer
[ ]Actinic Keratosis
[ ]Efudex
[ ]Seborrheic Keratosis
[ ]Allergic Contact Dermatitis

CONSULTS REVIEWED/ENTERED: BY MD n/a

RTC:
[ ] PRN
[ ]PENDING PATH
[X]OTHER 6 months

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at ALVIN C. YORK VAMC

Progress Notes

UNDERSTANDING OF ALL INSTRUCTIONS VOICED: YES
[X]Patient
[ ]Spouse
[ ]Caregiver

GOAL: COMPLIANCE WITH TREATMENT REGIMEN

OTHER NOTES: Veteran is to: stop hair relaxers, use hand lotion to condition hair rather than hair conditioner, incorporate 90 grams of protein into diet daily, return one morning for blood draw in th out-patient lab (cortisol level).

/es/ LISA D EARP
RN
Signed: 05/20/2015 14:59

05/20/2015 ADDENDUM STATUS: COMPLETED
Provided veteran with a bookmark list of phone numbers including direct number for Dermatology and the number for pharmacy. Above instructions were written down for veteran. She is aware a non formulary consult has been submitted for Biotin and if approved will be mailed to her. She can call the pharmacy in 24 to 48 hours to check on status.

/es/ LISA D EARP
RN
Signed: 05/20/2015 15:03

LOCAL TITLE: DERMATOLOGY OUTPATIENT CONSULT REPORT (030)
STANDARD TITLE: DERMATOLOGY CONSULT
DATE OF NOTE: MAY 20, 2015@14:38 ENTRY DATE: MAY 20, 2015@14:38:42
AUTHOR: ARZUBIAGA,CARMEN M EXP COSIGNER:
URGENCY: STATUS: COMPLETED

47 yr old bf seen in consultation for hair brakage and hyperpigmentation.dorsum of feet and hands> she admits to using relaxers> she denies traction, hair pieces

She was seen in Nashville 2009
" relates a story of hair loss that began at age 19. At first, her hair started to break off 3-4 inches from the root in the frontal scalp. At 22yrs, the breakage in the front became more diffuse. This lead the patient to cut all of her hair. The hair all grew out, but the breakage resumed in a tiny frontal spot for 5 years. Over the past 10 years, the breakage continued, with some improvement when she began taking synthroid. The original spot on the frontal scalp is still short and has not regrown. No relaxing treatments. Using Nexxus shampoo/conditioner twice weekly. Never had traction, glued in hair pieces or hairweaves. Not using grease. She

Progress Notes

says that she is sure that it is from hormone inbalances. She gets her periods regularly but she does not "ovulate" She also has hair growth on her chin.

She said that she has flaking but not in the area of the hair loss.

ALL:
Codeine
Shellfish

PMHx:
Denies NMSC, Melanoma, Abnormal moles
Regular menstrual cycles

FamHx:
Denies NMSC, Melanoma, Abnormal moles
Denies alopecia

SocHx:
Denies Tobacco, EtOH, IVDU
Unemployed. Single. Lives in Antioch, TN.

MEDS: reviewed in CPRS.
Atenolol
Synthroid
Ibuprofen

EXAM:
GEN: WDWN AAF, in NAD, AxO x3
SKIN: examined face, lips, eyelids, neck, scalp, BUE:
1. On the left frontal scalp is a 7x7cm patch of short (0.5cm) terminal hairs. Patching in appearance, no discrete areas of alopecia. Boggy in texture with some flaking. No scarring is visible.

Labs: Free Testosterone, DHEAS, 17-OH progesterone, prolactin, iron, TIBC, ferritin: WNL

Biopsy of scalp

A PAS stain was obtained but was unremarkable. The typical features of a tinea infection of the scalp are not appreciated. The biopsy demonstrates slightly decreased number of terminal hairs but there is no evidence of trichomalasia or internal root sheath trauma, features which would normally be expected with trichotillomania. Eosinophils and "swarm of bees" inflammatory infiltrates normally appreciated with alopecia areata are also not noted making the diagnostic considerations androgenetic alopecia, telogen effluvium or antigen effluvium.

DIAGNOSIS: SKIN, SCALP, PUNCH BIOPSY: MILD NON-CICATRICIAL ALOPECIA "

Progress Notes

O)hair split in the middle
no areas of alopecia
mild hyperpigmentation dorsum of feet and hands

A)hair breakage ? secondary to relaxers and traction (pt wears a ponytail)
R/o addisons disease

P)stop relaxers
wash with mild shampoo and conditioner
stop traction
trial of biotin 300mcg q day.
eat 90grms of protein a day.
cortisol am

/es/ CARMEN M ARZUBIAGA, MD
M.D
Signed: 05/20/2015 15:10

LOCAL TITLE: DERMATOLOGY GENERAL
STANDARD TITLE: DERMATOLOGY NOTE
DATE OF NOTE: MAY 20, 2015@14:02 ENTRY DATE: MAY 20, 2015@14:02:57
AUTHOR: ROMANS,MARTHA A EXP COSIGNER:
URGENCY: STATUS: COMPLETED

*** DERMATOLOGY GENERAL Has ADDENDA ***

Consult or Major Complaint:consult copied-Patient concerned of trichorrhexia and hyperpigmentation.

Last seen in Clinic:2009 Nash Derm-mild non-cicatricial alopecia

Are you taking ASA or ASA products? none

Are you taking any blood thinning medications? none

Do you have a pacemaker, artifical joints or valves? none

Are you allergic to lidocaine? denies allergy

Have you had fever (> 100.4 degrees F) in the past 24 hours? no

/es/ MARTHA A ROMANS
LPN

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at ALVIN C. YORK VAMC

Progress Notes

Signed: 05/20/2015 14:08

05/20/2015 ADDENDUM STATUS: COMPLETED
Active and Recently Expired Inpatient and Outpatient Medications (including Supplies):

Active Outpatient Medications	Status
1) LEVOTHYROXINE (SYNTHROID) 0.025MG=25MCG TAKE ONE-HALF TABLET BY MOUTH EVERY DAY BEFORE BREAKFAST - FOR THYROID -TAKE ON EMPTY STOMACH	ACTIVE

Copy of Medication Reconciliation Report was given to the patient to review before seeing the MD. [X]YES [ ] NO

/es/ MARTHA A ROMANS
LPN
Signed: 05/20/2015 14:09

Exhibit I

Department of Veterans Affairs
Tennessee Valley Healthcare System
Alvin C. York Campus
3400 Lebanon Pike, TN 37129-12367
www.tennesseevalley.va.gov

Dermatology **Clinic**

To speak to the clinic nurse regarding a medical question/concern

(615) 225-2873

To schedule, change, or cancel an appointment: (615) 225-3600 or
1 (800) 228-4973 ext. 23600

Telephone Care:
(to speak to a nurse after hours):
(615) 225-3700 or
1 (800) 228-4973 ext. 23700

Pharmacy/Prescription Refills:
(615) 321-6325 or
1 (866) 786-9367 ext 23705
Or online via My HealtheVet at
www.myhealth.va.gov

For Billing Inquiries:
(615) 225-3500 or
1-800-228-4973) Ext. 23500

Release of Information: (to obtain copies of your medical record):
(615) 225-2807 or
1-800-876-7093)

Suicide Hotline:
1 (800) 273-8255

10100

Exhibit I

Use hand lotion to condition hair rather than hair conditioner.

Stop hair relaxers.

You need 90grams of protein in your diet daily.

Return one morning for blood draw in the out-patient lab. (Cortisol level)

Return to Dermatology in 6 months

Exhibit 1

Report from: MURFREESBORO Station #626A4
05/19/2015 09:39

********* CONFIDENTIAL Med Recon Act/Pend/Exp/Non VA SUMMARY pg. 1 **********
WEST,KENDRIA Y

---------- PSO - Recent Rx Profile ----------

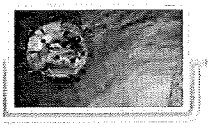
Allergies: CODEINE,
Adverse Reactions: _____ ,

Active and Recently Expired Outpatient Medications (including Supplies):

	Active Outpatient Medications	Status
1)	LEVOTHYROXINE (SYNTHROID) 0.025MG=25MCG TAKE ONE-HALF TABLET BY MOUTH EVERY DAY BEFORE BREAKFAST - FOR THYROID -TAKE ON EMPTY STOMACH	ACTIVE

*** END * CONFIDENTIAL Med Recon Act/Pend/Exp/Non VA SUMMARY pg. 1 *********

Biotin supplement
Non-formulary request submitted



Trichorrhexis nodosa

Definition

Trichorrhexis nodosa is a common hair problem in which thickened or weak points (nodes) along the hair shaft cause your hair to break off easily.

Alternative Names

Hair shaft fracture; Brittle hair; Fragile hair; Hair breakage

Causes

Trichorrhexis nodosa can be an inherited condition.

The condition may be triggered by things such as blow-drying, over-brushing, perming, or excessive chemical use.

In some cases, trichorrhexis nodosa is caused by an underlying disorder, including very rare ones, such as:

- Thyroid not making enough thyroid hormone (hypothyroidism)
- Buildup of ammonia in the body (argininosuccinic aciduria)
- Iron deficiency
- Menkes syndrome (Menkes kinky hair syndrome)
- Group of conditions in which there is abnormal development of the skin, hair, nails, teeth, or sweat glands (ectodermal dysplasia)
- Trichothiodystrophy (inherited disorder that causes brittle hair, skin problems, and intellectual disability)
- Biotin deficiency (inherited disorder in which the body is not able to use biotin, a substance needed for hair growth)

Symptoms

Your hair may break easily or it may appear like it is not growing.

In African Americans, looking at the scalp area using a microscope shows that the hair breaks off at the scalp area before it grows long.

In other people, the problem often appears at the end of a hair shaft in the form of split ends, thinning hair, and hair tips that look white.

Exams and Tests

The health care provider will examine your hair and scalp. Some of your hairs will be checked under a microscope or with a special magnifier used by skin doctors.

Blood tests may be ordered to check for anemia, thyroid disease, and other conditions.

Treatment

If you have a disorder that is causing trichorrhexis nodosa, it will be treated.

Your provider may recommend measures to reduce damage to your hair such as:

- Gentle brushing with a soft brush instead of aggressive brushing or ratting
- Avoiding harsh chemicals such as those used in straightening compounds and perms
- Not using a very hot hair dryer for long periods and not ironing the hair
- Using a gentle shampoo and a hair conditioner

Outlook (Prognosis)

Improving grooming techniques and avoiding products that damage hair will help correct the problem.

A.D.A.M. Navigator

- Encyclopedia Home
- Diseases A-Z
- Printer-friendly version

Penn State Hershey Services

- Dermatology
- Find a Physician

Images


Hair follicle an...

Read More

- Hair loss
- Broken bone

This condition is not dangerous, but may affect a person's self-esteem.

When to Contact a Medical Professional

Call your provider if symptoms do not improve with changes in grooming and other home-care measures.

References

James WD, Berger TG, Elston DM. Diseases of the skin appendages. In: James WD, Berger TG, Elston DM, eds. *Andrews' Diseases of the Skin*. 12th ed. Philadelphia, PA: Elsevier; 2016:chap 33.

Patterson JW. Diseases of cutaneous appendages. In: Patterson JW, ed. *Weedon's Skin Pathology*. 4th ed. Philadelphia, PA: Elsevier Churchill Livingstone; 2016:chap 15.

Review Date: 5/2/2017
Reviewed By: David L. Swanson, MD, Vice Chair of Medical Dermatology, Associate Professor of Dermatology, Mayo Medical School, Scottsdale, AZ. Also reviewed by David Zieve, MD, MHA, Medical Director, Brenda Conaway, Editorial Director, and the A.D.A.M. Editorial team.

Exhibit K

Protein Requirements - Dietary Reference Intakes

Inputs

Gender: female
Age (years): 47
Weight: Weight: 66.4 kg (146.1 pounds)

Selections made:

Analysis

Based on the entries above: Estimated Average Requirement (EAR): The average daily protein intake level that is estimated to meet the requirements of half of the healthy individuals in your age and gender group is: 43.8 grams/day.

RDA

Recommended Dietary Allowance (RDA): The average daily dietary protein intake level that is sufficient to meet the nutrient requirements of nearly all (97-98 percent) healthy individuals in your age and gender group is: 53.1 grams/day.

Comments if any:

Diagnosis

Hair loss in women isn't always as straightforward as it is in most men. In men about 90 percent of all cases are caused by hereditary male pattern baldness. In women, however, hair loss can be triggered by a multitude of conditions and circumstances.

The below battery of diagnostic tests should be performed when attempting to pinpoint the hair loss trigger. These tests can at the very least eliminate the possibility of certain disorders causing your hair loss and perhaps aid in finding the actual cause. The truth of the matter is that for many patients these test usually come back with a reading of "within the normal range," but it's important to remember that the proper diagnosis of female hair loss usually starts of with the process of elimination.

Diagnostic Tests

Hormone levels (DHEAs, testosterone, androstenedione, prolactin, follicular stimulating hormone, and leutinizing hormone)

Serum iron

Serum ferritin

Total iron binding capacity (TIBC)

Thyroid stimulating hormone (T3, T4, TSH)

VDRL (a screening test for syphilis)

Complete blood count (CBC)

Scalp biopsy
A small section of scalp usually 4mm in diameter is removed and examined under a microscope to help determine the cause of hair loss.

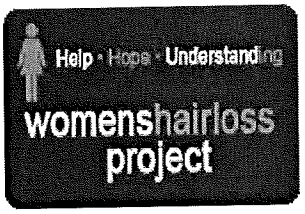
Hair pull
The hair pull test is a simple diagnostic test in which the physician lightly pulls a small amount of hair (approx 100 simultaneously) in order to determine if there is excessive loss. Normal range is one to three hairs per pull.

Densitometry
The densitometer is a handheld magnification device which is used check for miniaturization of the hair shaft.

Reviewed by Paul J. McAndrews, MD

Hair Loss Message Boards

Visit The Hair Loss Forum



Women's Hair Loss Project

Exhibit M

HIV TEST RESULTS Neg
VERIFIED BY HM3 [illegible]
CMD UIC 68470 DATE 2-14-89
ROSTER 007 MTE PACOM

OTHER IMMUNIZATIONS

	DATE	TYPE	DOSE	REACTION	REMARKS	PHYSICIAN'S NAME
1	14 MAR 1988	MENINGOCOCCAL VAC.	0.5ml		G.E. COY, CDR, MC, USNR	
2	14 MAR 1988	INFLUENZA	0.5ml		G.E. COY, CDR, MC, USNR	
3	21 MAR 1988	ORAL POLIO TRIVALENT 1	0.5ml		G.E. COY, CDR, MC, USNR	
4		ORAL POLIO TRIVALENT 2	0.5ml			
5	14 MAR 1988	ADENO VIRUS 4 & 7	2 TABS		[illegible] CDR, MC, USNR	
6		RUBELLA	0.5ml			
7		BICILLIN	1,200,000u			
8	21 MAR 1988	RUBEOLA			G.E. COY, CDR, MC, USNR	
9	21 Aug 86	Plague #1	1.0cc			[illegible]
10	22 Dec 86	PLAGUE #2	0.2cc			HD PACIO
11	OCT 27 1987	INFLUENZA	0.5cc			[illegible] HM3
12	OCT 27 1987	HEPAVAX #1	1cc			[illegible]
13	NOV 23 1987	Hepavax #2	1.0cc			[illegible]
14	[illegible]	Hep B #3	1cc			HM3 [illegible]
15	27 OCT 88	FLU VAC	.5cc			S. DICKERSON HM3

SENSITIVITY TESTS (Tuberculin, etc.)

	DATE	TYPE	DOSE	ROUTE	RESULTS	PHYSICIAN'S NAME
1	18 MAR 1988	PPD-S 0.0001mg	.1ml	INTRADERMAL	error ~~INDURATION~~ G.E. COY,	CDR, MC, USNR
2		PPD Converter	— Do Not		Give	
3	[illegible] 87	PPD	1 TU	I.D	15 mm	Michael [illegible]
4	OCT 27 1987	HIV ANTIBODY SCREENING			NEG	[illegible]
5						
6						
7						
8						
9						
10						

REACTIONS (To transfusions, drugs, sera, foods, allergens, etc.)

	DATE	AGENT	TYPE OF REACTION	SEVERITY	PHYSICIAN'S NAME
1					
2					
3					
4					
5					

BLOOD TYPING

	DATE	TYPE (International)	Rh FACTOR	PHYSICIAN'S NAME
1	17 MAR 1988	O	POS	
2				G. E. COY, CDR, MC, USNR
3				

REMARKS AND RECOMMENDATIONS (Including history of diseases for which any of the above immunizing agents were given with year and place of attack)

07 APR 1986 HTLV III NEG BY ELISA CDR COY

THIS RECORD IS ISSUED IN ACCORDANCE WITH ARTICLE 99, WHO SANITARY REGULATION NO. 2

RECORD BEFORE THE AGENCY (RBA)

Exhibit M

DATE	SYMPTOMS, DIAGNOSIS, TREATMENT, TREATING ORGANIZATION *(Sign each entry)*
JUL 23 1987	STAFF SICK CALL NAVAL HOSPITAL SAN DIEGO SAN DIEGO, CA 92134 – No Known Medicinal Allergies –
(9) B/P: 112/60	19 y/o female present to S S C
(9) pulse 72	to FU PPD Reactor
temp 95.8	Pt states she has been PPD converter since 1983
	at ~~which time~~ which time ~~was~~ received ī yr
	course of INH. Denies ~~was~~ hung Fever chills night
	sweats, SOB, has had nl CXR in past (83), HIV ⊖ in 1986
	O – AF VSS
	chest: clear.
	A/P ↓ PPD converter since 1983, tx c̄ INH x 1 yr in 1983
	– ✓ CXR
	– if negative will give ITU PPD.
	– Return for clinic ~~in~~ Monday [signature]

★ U.S. Government Printing Office: 1986—491-248/40085

STANDARD FORM 600 BACK (REV. 5-84)

RECORD BEFORE THE AGENCY (RBA)

00442

2

108

Exhibit M

DATE	SYMPTOMS, DIAGNOSIS, TREATMENT, TREATING ORGANIZATION *(Sign each entry)*
JUL 31 1987	STAFF SICK CALL NAVAL HOSPITAL SAN DIEGO SAN DIEGO, CA 92134 — No Known Medicinal Allergies —
(S) B/P: 104/72	f/u
(oral) Temp: 98.0	S – 19 y.o. Black ♀ presents to SSC for f/u hx of
(S) Pulse: 74	PPD conversion.
	19 y/o B/♀ c̄ ⊕ PPD in 1983
	∇ was treated for a full year c̄ INH.
	Pt denies CP, cough, febrile illness recently.
	Pt was tested c̄ 1 TU PPD on July 27/87
	and now has > 5 mm induration.
	A: ⊕ PPD 1983; Full [illegible] INH; 1 TU ⊕ now
P:	① Send to preventive medicine today
	② Pt recommended to return [illegible] for checkup
	Michael Torricelli TORRICELLI, M. R. MC [illegible] [redacted] USN

U.S. Government Printing Office: 1986—491-248/40088 STANDARD FORM 600 BACK (REV. 5-84)

RECORD BEFORE THE AGENCY (RBA)

00440

3 109

11. Hair relaxing
    - Hair relaxing is weakening to the hair shaft, but can actually facilitate hair length in patients with kinky hair. This is due to decreased hair breakage during combing. The relaxing procedure straightens the hair and makes it easier to groom, but the grooming should be done gently to avoid hair shaft fracture.

12. Hair permanent waving
    - Lastly, hair permanent waving is also damaging. The curls should be as loose as possible with the interval between procedures as long as possible. For patients with damaged hair, the perming solution should be weak and left in contact with the hair for as short a period as possible.

Shampoos and Hair Products

- Telogen Effluvium
  - Focus on hairspray for body and not stiffness
  - Gel
- Seborrheic Dermatitis
  - Head and shoulders – good for mild seb derm, good for prolonging disease-free episodes. Good technology
  - Nizoral 2% – effective, but can be very drying. Use with conditioners
- Women of Color
  - Pantene brown bottle
  - Keracare
- Colored hair
  - A color protectant shampoo and conditioner such as L'Oreal Vive
  - L'Oreal Vive (shampoo and conditioner) or other UV protectant
- Fine hair
  - AVOID Pantene
  - Use dimethicone-based products → will smooth cuticle, hair is shiny, looks more voluminous

Progress Notes

AUTHOR: LIVINGOOD,MATTHEW R EXP COSIGNER: STRICKLIN,GEORGE P
URGENCY: STATUS: COMPLETED

*** DERMATOLOGY CLINIC GENERAL Has ADDENDA ***

PROGRESS NOTE:

Chief Complaint: f/u hair loss

S: Mr. West is a 47 yo F w/ PMH of hashimoto's thyroiditis and chronic hair loss who presents for f/u of chronic L frontal hair loss. Pt reports that her her loss started when she was in the military ~20-30 yrs ago w/o precipitating event or trauma. Pt noticed that her hair would break off a few inches from the root of her hair on the L frontal scalp. A few years later, the breakage became slightly more diffuse, but still localized to the L frontal hair line. Pt reports that she was diagnosed with Hashimoto's thyroiditis in 2004 and started on levothyroxine thereafter, but w/ no improvement in hair loss in affected region. Pt reports that she continues to experience hair loss in the L frontal scalp and is frustrated that a cause cannot be found. Punch bx of frontal scalp taken in 2009 and revealed:

Microscopic exam/diagnosis:
A PAS stain was obtained but was unremarkable. The typical features of a tinea infection of the scalp are not appreciated. The biopsy demonstrates slightly decreased number of terminal hairs but there is no evidence of trichomalasia or internal root sheath trauma, features which would normally be expected with trichotillomania. Eosinophils and "swarm of bees" inflammatory infiltrates normally appreciated with alopecia areata are also not noted making the diagnostic considerations androgenetic alopecia, telogen effluvium or antigen effluvium.

DIAGNOSIS: SKIN, SCALP, PUNCH BIOPSY: MILD NON-CICATRICIAL ALOPECIA (SEE MICROSCOPIC).

Bx findings did not suggest any scarring process and possible dxs were listed as androgenetic alopecia, telogen effluvium, or anagen effluvium.

Pt currently repors using Nexus condtioner every 4 days along with unspecified baby shampoo every 4 days as well to hair. Utilizes occasional coconut oil and also reports using chemical relaxers every few months to her hair (last relaxer tx was 6 wks ago per pt). Pt denies hx of using severe traction of hair, glued in hair pieces, or hairweaves. Pt does not apply grease to scalp. Lab workup in the past has not been suggestive of metabolic or hormonal abnormalities responsible for hair loss, but pt is adamant that this is the likely cause.

Pt has attempted tx w/ biotin and increased protein consumption in the past with no improvement. She does not wish to restart biotin today and would like her hair examined under a microscope. Pt is overall very frustrated with her hair loss and would like someone to help her.

Progress Notes

PMH, family hx, social history: reviewed. Notable for:
- lives in Nasvhille, TN
- single
- unemployed
- no FHx of hair loss or autoimmune disease

ROS: no fevers, chills, skin pain; skin per HPI

Medications: reviewed in CPRS
All outpatient medications have been reviewed with the Patient, Family and/or Caregiver as medically possible. The Patient, Family and/or Caregiver has been provided an updated copy of the Outpatient Medication List. The patient was informed of his/her responsibility to maintain a current medication list and communicate information to all providers.

Allergies: reviewed in CPRS

Exam:
Gen: NAD, normal mood and affect
Skin: Head, neck, scalp, eyelids, lips, chest, back, abdomen, bilat UE and bilat LE examined (declined add'l aras of exam):

- Over L frontal forehead: there is a ~4 x 4 cm annular area of hair w/ normal hair density and color w/ mid-hair breakage; no scarring; no perifollicular scaling or erythema; no papules or pustules noted today on PE

- remainder of scalp w/o focal or diffuse hair loss

- no irregularities of hair under light microscopy today on exam

Labs:
- CBC, CMP, lipids (except LDL 112), TFTs wnl (2/7/15)
- vit B6 wnl (2/6/15)
- 17 hydroxy progesterone, DHEAS, free testosterone, ferritin, prolactin, iron, TIBC wnl (9/9/2009)
- FSH, LH, B12, folate wnl (8/12/15)

A/P:
1. Non-scarring alopecia of unknown etiology: most likely due to truama or traction. Skin bx in the past has ruled out alopecia areata, inflammatory/infection, and scarring alopecia. Light microscopy of hair shaft reveals no irregularities on exam today. Lab workup in past has ruled out sufficient causative nutritional causes. Pt does have hashimoto's thyroiditis, but her thyroid hormone appears to be appropriately augmented with levothyroxine given TFTs from 2/15. Pt additionally on no medications that are commonly associated w/ focal hair loss.
- pt encouraged to use regular conditioner and mild shampoo
- encouraged to stop chemical relaxer txs

- pt encouraged to utilize hair styles that place minimal traction on affected area of hair loss
- pt declines interest in augmentation w/ PO biotin
- of note, pt was combative during clinic visit and appeared fixated on thought that there is metabolic/systemic cause responsible for her focal hair loss. Pt believes that she has tricorrexis nodosa as well given her personal reading. Pt was generally unwilling to listen to hypothesis that focal hair loss could be due to traction and manipulation

RTC prn as pt reports that she no longer wishes to be followed by NASH-VA dermatology

Pt left dermatology clinic in no pain or discomfort.

Dr. Sticklin is the attending of record for this encounter.

/es/ MATTHEW ROSS LIVINGOOD, MD
Clinical Fellow
Signed: 09/17/2015 17:36

/es/ GEORGE P STRICKLIN, MD
Chief of Dermatology/NASH
Cosigned: 09/21/2015 08:48

09/21/2015 ADDENDUM STATUS: COMPLETED

I saw and evaluated Ms. West and concur with all of the resident's note. History and laboratory findings as noted. Examination reveals normal density hair growth on scalp, no erythema, induration or scarring of the scalp. The left frontal hairline is notable for mid-hair breakage with about 2 cm of growth. The scalp hair is flattened consistent with the use of relaxer about 6 weeks prior per patient report.

This pattern is most consistent with trauma to the hair. Her endocrine history and focus upon this is noted but the normal density, thickness and focal nature of the breakage is not consistent with an endocrine cause. One might consider an underlying mosaic/nevoid condition but those typically appear in childhood and the skin biopsy was unrevealing.

I agree with the recommendations to avoid trauma to the area. This would include the use of relaxers, permanents, bleaches, hot oil, braiding/weaves. Given the focality of the problem, manipulation of the hair may also be included in this listing. All of these weaken the hair and may contribute to breakage.

Ms. West was highly focused upon an endocrine etiology of this focal hair loss and did not seem to be receptive of the medical conclusions and advice. She was assured that a comprehensive medical, pathological and laboratory workup of this condition did not reveal any specific etiology. Fortunately, there was also no evidence of scarring. She appeared quite displeased with our conclusions and advice.

Exhibit O

Progress Notes

Printed On Oct 8, 2015

Followup at MU-Derm as needed.

/es/ GEORGE P STRICKLIN, MD
Chief of Dermatology/NASH
Signed: 09/21/2015 09:12

[redacted]

PATIENT NAME AND ADDRESS (Mechanical Imprinting, if available)
WEST,KENDRIA Y
1109 BLUE WILLOW CT
ANTIOCH, TENNESSEE 37013
[redacted]

VISTA Electronic Medical Documentation
Printed at MURFREESBORO

ELSEVIER
SAUNDERS

Crit Care Clin 21 (2005) S1–S8

CRITICAL
CARE
CLINICS

Unmasked Adult-Onset Urea Cycle Disorders in the Critical Care Setting

Marshall L. Summar, MD[a,*], Frederick Barr, MD, MS[b,c], Sheila Dawling, PhD[d], Wendy Smith, MD[e,f], Brendan Lee, MD, PhD[g], Rani H. Singh, PhD, RD[h], William J. Rhead, MD, PhD[i], Lisa Sniderman King, MSc[j], Brian W. Christman, MD[h,k]

[a]*Center for Human Genetic Research, Division of Medical Genetics, Department of Pediatrics, Vanderbilt University Medical Center, Nashville, TN, USA*
[b]*Department of Pediatrics, Division of Pediatric Critical Care Medicine, Vanderbilt Children's Hospital, Nashville, TN, USA*
[c]*Department of Pediatrics, Vanderbilt University Medical Center, Nashville, TN, USA*
[d]*Associate Professor of Pathology, Department of Pathology, Vanderbilt University Medical Center, Nashville, TN, USA*
[e]*Division of Genetics, The Barbara Bush Children's Hospital, Maine Medical Center, Portland, ME, USA*
[f]*Department of Pediatrics, Division of Genetics, Maine Pediatric Specialty Group, Portland, ME, USA*
[g]*Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, TX, USA*
[h]*Division of Human Genetics, Emory University School of Medicine, Atlanta, GA, USA*
[i]*Medical College of Wisconsin Genetics Center, Madison, WI, USA*
[j]*University of Washington, Seattle, WA, USA*
[k]*Division of Pulmonary and Critical Care Medicine, Department of Medicine, Vanderbilt University Medical Center, Nashville, TN, USA*

Urea cycle disorders (UCD) are caused by a defect in the waste nitrogen disposal system that converts ammonia to urea (Fig. 1) [1–4]. A defect in or absence of one of the five enzymes, two substrate transporters, or cofactor producer of the cycle impedes the conversion of nitrogen and its elimination as urea, often resulting in hyperammonemia.

The classic presentation of UCD has been critically high blood levels of ammonia in the newborn period [4–8]. This pattern constitutes most of the literature on the subject; however, there is a growing body of knowledge concerning urea cycle disorders that have manifested or only been recognized in adulthood. A partial or milder enzyme deficiency can permit an individual to function relatively normally, sometimes for decades, until confronted by an environmental stressor that triggers a hyperammonemic crisis. Box 1 lists some of the conditions described in the literature that can result in hyperammonemia in older patients. These conditions typically either increase the need for nitrogen clearance or interfere with the enzymes of the urea cycle. The underlying urea cycle disorder can be difficult to recognize, because the patient is frequently ill for other reasons. Nevertheless, prompt recognition is critical for treatment to be effective. Without early diagnosis and aggressive intervention, the prognosis for these patients is poor.

This article presents three cases of adult onset urea cycle disease precipitated by stressful medical situations in the intensive care environment. These cases have similarities in presentation and history that should provide clues for recognizing patients in similar situations. The report also outlines an approach to the diagnosis and management of adult patients who have hyperammonemia, which is different to some degree from suggested practice in hyperammonemia and suspected urea cycle disease in childhood.

Case 1

A white male in his early 30s presented following a four-wheeler accident. He had a right clavicular fracture and a right tibial plateau fracture requiring open reduction and internal fixation.

On the night following the orthopedic surgical procedure, he became confused and combative. He had tachycardia, hypertension with a diastolic pressure of 140 mmHg, and hyperthermia reaching a maximum of 106°. Additionally,

Dr. Summar acknowledges the support of NIH grants MOI-RR-0095 and U54-RR-019453.

Complete financial disclosure information for each author is provided in the frontmatter of this supplement on page iii.

* Corresponding author. Department of Pediatrics, Vanderbilt University Medical Center, 525D Light Hall, Nashville, TN 37232–0165.
E-mail address: marshall.summar@vanderbilt.edu (M.L. Summar).

doi:10.1016/j.ccc.2005.05.002

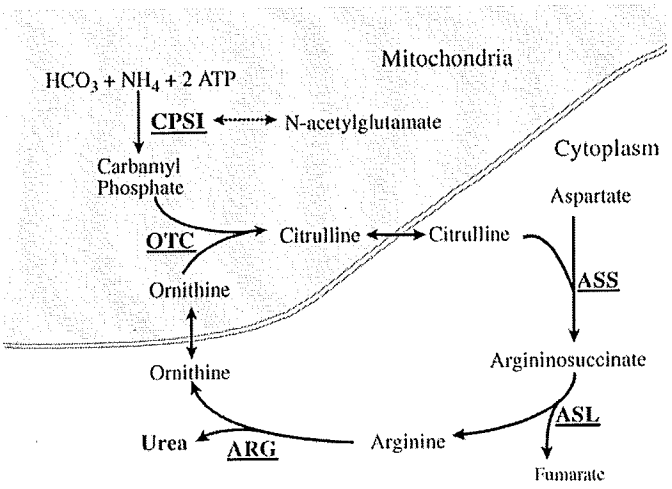


Fig. 1. Urea cycle pathway. ARG, arginase; ASL, argininosuccinic acid lyase; ASS, argininosuccinic acid synthetase; CPS1, carbamyl phosphate synthetase 1, OTC, ornithine transcarbamylase.

the patient exhibited generalized tonic-clonic seizures that the local hospital was unable to control.

Past medical history

Following a closed head injury at 5 years of age, the patient developed a seizure disorder that was well-controlled on carbamazepine and primidone. He had chronic hypertension, for which he was prescribed quinapril, amlodipine, and hydrochlorothiazide, although his compliance was questionable. His gastroesophageal reflux was being treated with pantoprazole, and he had a history of persistent anemia, with a hemoglobin of 9 and hematocrit of 27. Discussions with the patient's family revealed that he was an "auto-selective" vegetarian; he systematically picked out and rejected the meat in any dish served him. He also had a history of nonspecific psychiatric problems.

Box 1. Published reports of triggers of urinary cycle disorders in adulthood

Valproic acid [9–20]
Postpartum stress [21]
Heart-lung transplant
Short bowel and kidney disease [22]
Parenteral nutrition with high nitrogen intake [23,24]
Gastrointestinal bleeding [25]

Note that hyperammonemia, although not necessarily UCD, has also been reported in patients on high-dose chemotherapy, particularly during bone marrow transplantation, and in severe hepatic disease [26–28].

The patient had only an eighth-grade education and was described as "slow." He had one daughter, who was healthy.

Clinical course

CT evaluation at the local hospital showed what appeared to be a lacunar infarct of the left caudate. MRI indicated increased signal in the left frontal periventricular region; it was uncertain whether this was attributable to an old infarct or previous trauma. The patient's electroencephalograph EEG showed severe brain dysfunction in the setting of a seizure disorder. In fact, he remained in status epilepticus for 12 hours despite the administration of antiepileptics, and required intubation before transportation to our center.

Laboratory work at the outside hospital revealed a low blood urea nitrogen and an ammonia level of 553 μmol/L at approximately 24 hours into the course of coma.

Upon arrival at our tertiary care center, the patient was dialyzed to remove excess ammonia, and the level dropped quickly to 250 μmol/L. Despite this improvement, he remained in a deep coma and unresponsive to all stimuli. His pupils were fixed and dilated, and remained so during his entire hospitalization. He displayed evidence for generalized cerebral edema. He was dialyzed further and placed on continuous veno-venous hemodialysis. He was begun on the nitrogen scavengers sodium benzoate and sodium phenylbutyrate, the latter solubilized and placed in the gastrointestinal tube. The patient also received arginine hydrochloride and was started on a glucose insulin drip in an effort to prevent further catabolism.

Despite aggressive therapy, the patient's ammonia level never fell below 200 μmol/L. A plasma amino acid profile showed very high glutamine and high alanine levels, as well as very low citrulline and low arginine concentrations. On

the second hospital day, the patient developed hypotension, requiring norephinephrine, and progressive hypoxia, despite being on a ventilator and receiving aggressive ventilatory management. He subsequently developed pulmonary edema and failed an apnea test. After extensive discussion with his family, support was withdrawn, resulting in rapid asystole and death.

Diagnosis

The presumptive diagnosis was hyperammonemia secondary to a urea cycle disorder, possibly ornithine transcarbamylase (OTC) or carbamyl phosphate synthetase (CPS1) deficiency. Based on a liver biopsy with enzymatic testing and DNA sequence analysis, the patient had partial N-acetylglutamate synthetase (NAGS) deficiency and was unable to make cofactor for the CPS enzyme (see Fig. 1).

Case 2

A 58-year-old white female who had a history of recurrent episodes of "asthmatic bronchitis" presented to her local hospital having severe wheezing of several days' duration and a productive cough. She was thought to have a viral illness that was exacerbating her asthma. She was treated with an aerosol bronchodilator, but continued to have bronchospasm and dyspnea with wheezes.

The patient was admitted to the outlying hospital and started on intravenous steroids (methylprednisolone), antibiotic therapy (cefotaxime) and intravenous fluids. She was also receiving albuterol and loratadine/pseudoephedrine. Radiographs demonstrated no infiltrates, but the patient was maintained on antibiotics because of her age. Oral intake was minimal during this time. On day 5 of admission she developed acute confusion and an expressive aphasia with focal deficits, progressing to a complete coma within 48 hours. A work-up, including lumbar puncture, EEG, two head CTs, a carotid Doppler test, and a cerebral three-vessel arteriogram, was nondiagnostic. The patient was listed as having steroid psychosis. She was also begun on acyclovir against the possibility of herpes encephalitis, and transferred to the critical care unit at our institution.

The patient was intubated, exhibiting decorticate posturing and sluggish pupils, but no seizures. Her temperature and pulse were normal, blood pressure was 142/80 mmHg, and toes were upgoing in the Babinski reflex.

Past medical history

The patient had recurrent episodes of asthma, along with allergic rhinitis and nasal polyps. She had undergone numerous procedures, including a cholecystectomy, appendectomy, hysterectomy, and a thyroid nodule treated with radiation; however, she had never received intravenous steroids (a critical point in understanding her case). According to her husband, she was on a regular diet, consumed a normal amount of food with a normal protein intake, and had no history of seizures or lethargy. Subsequent history from the patient revealed that she tended to avoid large protein intakes.

Clinical course

The diagnostic and laboratory workup revealed an ammonia level of 120 μmol/L at first measurement, later rising as high as 280; cerebral edema on CT examination; diffuse slowing but no seizure activity on EEG; and very low citrulline and arginine, with a mild elevation of glutamine per her plasma amino acid profile.

The patient was started on dialysis, intravenous sodium phenylacetate/sodium benzoate (after obtaining informed consent), arginine supplementation, and increased intravenous calories as dextrose.

The patient awoke within 8 hours of the initiation of dialysis and drug therapy and was discharged 3 days later. Follow-up over 5 years showed no residual deficits. Despite the earlier cerebral edema, visual field examination has shown no evidence of optic nerve crush injury. Intravenous steroids have been avoided, although this has been difficult in the face of the patient's worsening asthma. Now in her 60s, she has also developed mild pulmonary hypertension. She has not required dietary modification or oral nitrogen scavenging drugs (sodium benzoate, phenylbutyrate).

Diagnosis

Based on family history elicited from the patient's husband, the patient was diagnosed as a symptomatic OTC carrier (ornithine transcarbamylase is on the X chromosome). She had had four male infants who died from OTC deficiency. She has two adult OTC defect-carrier daughters. One (a normal college graduate) lost a son. The other has mild mental retardation and has suffered several mild hyperammonemic episodes in the past.

Case 3

A 34-year-old morbidly obese female was admitted to an outlying hospital 8 months after gastric bypass surgery with a Roux-en-Y procedure. Her presenting symptom was weakness, which progressed rapidly to uncontrolled status epilepticus and neurologic unresponsiveness requiring intubation.

The patient's serum ammonia concentration was 442 μmol/L and her initial aspartate aminotransferase level was elevated, although there was no evidence of transaminitis subsequently. Lumbar puncture and all cultures were negative, except for methicillin-resistant *Staphylococ-*

cus aureus in the sputum. She was placed on broad-spectrum antibiotics without improvement, and transported to our tertiary center.

Past medical history

The patient had been morbidly obese, having previously weighed 400 lbs, which was reduced to 160 lbs during the 8 months after her bariatric surgery. She had a history of respiratory distress syndrome and pneumonia during this period. She had a history of dumping syndrome with hypokalemia and hypernatremia. She also had non insulin-dependent diabetes mellitus and nephrolithiasis. She had been almost chronically hospitalized after her bariatric surgery, but no attempt was made to reverse or modify the procedure. Questioning revealed that she tended to avoid protein, preferring starch and carbohydrate. She had a history of depression.

Clinical course

At our center, the patient's CT examination was normal at first, subsequently exhibiting some cerebral edema. Lumbar puncture showed no culture growth for either viral, fungal, or bacterial organisms. An abdominal CT indicated possible fatty liver, and her chest radiograph revealed an infiltrate in the left lower lung suspicious for pneumonia. At this point she had been in status epilepticus for over 24 hours.

The patient was started on dialysis, phenylbutyrate, and citrulline. Her ammonia levels dropped to less than 100 μmol/L. An organic acid profile showed no orotic acid and an elevated 5-oxoproline and lactic acid.

In spite of the reduction in ammonia, which ultimately decreased to 21 μmol/L, the patient did not regain consciousness, even after 6 days of therapy. Following extensive consultation with her family, the patient was electively extubated and expired within 3 to 4 hours.

Diagnosis

Autopsy and liver biopsy were performed at the time of death. The liver biopsy demonstrated CPS1 deficiency and subsequent mutation analysis uncovered a defect consistent with that finding. In addition, fatty liver infiltrate and a large number of kidney stones were observed at autopsy.

Discussion

These three cases share some common clinical features. They all demonstrated a dramatic and rapid increase in nitrogen load, whether from trauma, rapid weight loss, or increase in protein turnover from intravenous steroid (prednisolones). All three patients presented having altered mental function that progressed to a markedly obtunded state and eventually coma. Their findings were consistent with toxicity from ammonia elevation, and consistent with that seen in patients who have both urea cycle disorders and chronic liver disease. The patient who had multiple orthopedic fractures had to deal with the breakdown and processing of the blood that was lost into the tissues surrounding the breaks. This can often amount to several units of blood, metabolism of which releases a large amount of waste nitrogen. The bariatric surgery patient suffered from malabsorption and nutritional disruption. The rapid weight loss resulted in protein catabolism in her tissues and excess nitrogen. The intravenous steroids used in the asthmatic patient resulted in a generalized increase in protein turnover, which also resulted in excessive nitrogen release [29].

The three patients also exhibited rapid deterioration of neurologic status, with the severity of their encephalopathy masked by their ongoing medical condition. The comorbid conditions led to delays in diagnosis. There was some evidence for cerebral edema by clinical examination or radiograph in all three patients, and seizures occurred in two of the three cases. Because they had all been quite ill and hospitalized, there was a decrease in oral intake leading up to and contributing to the decompensation.

Consistent with the literature regarding unmasked adult cases of UCD, the underlying molecular defects in these patients were at the beginning portion of the urea cycle. These defects tend to be more severe, because blockade in these steps occurs before any nitrogen is pulled into the cycle intermediates (see Fig. 1).

Reaching hyperammonemic threshold

In patients like these who manage to survive to adulthood before reaching a hyperammonemic threshold, the physiologic mechanisms that can tip the nitrogen balance appear to break down into three categories: (1) nitrogen turnover and nitrogen load from catabolism or sudden protein processing, outlined in Box 2; (2) diminished access to processing in the liver, the target organ for most nitrogen clearance, outlined in Box 3; and (3) the genetic capacity of the urea cycle to handle the nitrogen load, outlined in Box 4.

In turn, each of these is affected by a variety of factors.

Alterations in nitrogen turnover and load can be triggered by poor nutritional intake and rapid weight loss, such as that associated with gastric bypass surgery, chronic starvation, or self-starvation disorders such as anorexia and bulimia. They might also be precipitated by internal bleeding or damage, whether resulting from a large bone fracture or surgical trauma, or by viral illnesses and other chronic diseases that cause total body stress. Even the postpartum period has been implicated as a trigger mechanism [21].

A dramatic increase or decrease in habitual protein intake may also contribute to nitrogen imbalance. Consider high protein diet strategies, change in food access or preparation,

Box 2. Urea cycle defect triggers from nitrogen turnover and load

Rapid weight loss and poor nutritional intake
- Gastric bypass surgery

Internal bleeding or damage
- Fracture
- Surgical damage

Viral illness or other generalized stress

Postpartum period

Dramatic increase/decrease in habitual protein intake
- High protein diet strategies
- Change in food access or preparation
- Malabsorption conditions

Medications affecting protein catabolism
- Intravenous or high-dose glucocorticoids
- Chemotherapy

malabsorptive conditions, or medications that affect protein catabolism. Medications influencing protein turnover include high-dose or intravenous glucocorticoids, as well as chemotherapy. Most chemotherapy patients decrease their oral intake because of nausea or esophageal ulceration. Chemotherapy may also destroy some of the villi cells in the gut, thus compromising protein absorption. Elevations in ammonia and even a few deaths have been reported as a result of hyperammonemia from chemotherapy [26].

If access to functional hepatocytes is blocked, a physical urea cycle defect is created. This could occur following portacaval shunt (for severe cirrhosis), or varicocele shunting from a cirrhotic liver. Even a partial shunt could unmask a partial urea cycle disorder. So too could a decrease in the available hepatocyte pool. The most common cause of this decrement in liver function and resultant hyperammonemia is chronic cirrhosis, although acute chemical or viral damage could also be the underlying cause.

Box 3. Urea cycle defects triggered by decreased access to processing in liver

Vascular shunting of blood from liver
- Portacaval shunt
- Varicocele shunting from cirrhotic liver

Decrease in available hepatocyte pool
- Chronic cirrhosis
- Acute chemical or viral damage to the liver

Box 4. Genetic predisposing conditions affecting capacity of the urea cycle

Genetic defects in enzyme/transporter function of the urea cycle components or decreased function polymorphisms

Chemical or toxic affect on enzyme function

5-pentanoic acid (Jamaican vomiting sickness)

Valproic acid

Chemotherapeutic agents (cyclophosphamide)

Comorbid metabolic conditions
- Organic acidemias
- Fatty acid oxidation defects

Abnormalities in the urea cycle can stem from either genetic defects in the enzymes or transporters, or chemical/toxic effects on enzyme function. The classic toxicity affecting enzyme performance was 5-pentanoic acid (Jamaican vomiting sickness), although this is rarely seen anymore. Today the most widely recognized agent in this regard is valproic acid, which has a direct inhibitory affect on enzyme function [10,11,30,31]. There is some anecdotal evidence that zonisamide can also cause ammonia elevation. There is evidence of direct effects exerted on urea cycle function by chemotherapeutic agents, most notably cyclophosphamide [32,33]. Finally, metabolic comorbidities such as organic acidemias and fatty acid oxidation defects generate metabolites that can disrupt the urea cycle and potentially unmask a partial defect.

Diagnostic clues

There are clues suggestive of a possible UCD—illustrated to one degree or another in these cases—that can help the clinician recognize a urea cycle disorder. Although a family history of metabolic disease is a strong indicator, most families may not be aware of the existence of metabolic abnormalities, and the recessive inheritance pattern of most of these diseases may make the patient the only case. In older patients who had childhood symptomatology, the relatives familiar with the history may no longer be available, and the adult patient may not remember the childhood stories. A directed history is very useful. Specific questions should be asked about infant mortality, consanguinity, and other family characteristics. A dietary history of autoselective vegetarianism (ie, elective decreased protein intake), as well as high carbohydrate intake and obesity are other suggestive clues. A history of behavioral and psychiatric illness (possibly resulting from chronic low-grade hyperammonemia) or a history of prolonged clinical courses with seemingly routine illnesses should also prompt suspicion. For

example, past episodes of flulike illness in two of these patients created a prolonged illness, with lethargy and anorexia. If any of these historical points is present in an obtunded patient or if the clinician has a suspicion of a metabolic abnormality, an ammonia level should be ordered. If the ammonia is elevated, a metabolic specialist should be consulted and an amino acid profile should be obtained promptly, along with a repeat ammonia level.

Emergency management suggestions

Management of adult patients in a urea cycle-based hyperammonemic coma is predicated on three interdependent principles: first, physical removal of the ammonia by dialysis or some form of hemofiltration; second, reversal of the catabolic state through caloric supplementation and possible hormonal suppression; and third, pharmacologic scavenging of excess nitrogen.

Central venous access should be established and dialysis, if available, begun at the highest available flow rate as soon as possible. Dialysis is the most effective means of ammonia removal, and the rate is dependent on the flow through the dialysis circuit. In severe cases of hyperammonemia, provision for hemofiltration following the dialysis should be made until the patient is stabilized and the catabolic state is reversed. Some patients will reaccumulate ammonia after their initial round of dialysis and may require additional runs. Most patients will have a slight rise in ammonia after dialysis because removal by scavengers and the liver will not be as effective. This slight rise usually does not necessitate repeat dialysis.

The importance of managing the catabolic state is often overlooked. Because the catabolism of protein stores is often the triggering event for hyperammonemia, the patient will not completely stabilize until it is reversed. All three of the patients in this report suffered from catabolism, primarily because of reduced caloric intake and physiologic stress. Fluids, dextrose, and fat emulsion (Intralipid) should be given to blunt the catabolic process. The patient should be assessed for dehydration and fluids replaced. Because these patients suffer from cerebral edema, care should be taken to prevent overhydration. The nitrogen scavenging drugs are usually administered in a large volume of fluid, which should be taken into consideration. A regimen of 80 cal/kg/d is a reasonable goal. Although it is not common practice at all centers, the administration of insulin and glucose to artificially suppress the catabolic state is useful in profound catabolic states. This should only be done under carefully controlled conditions. At the same time, protein should be temporarily removed from intake (by mouth or total parenteral nutrition). Supplementation of arginine serves to replace arginine not produced by the urea cycle (in addition to the partial cycle function it can stimulate), and prevents its deficiency from causing additional protein catabolism. Refeeding the patient as soon as practicable is useful, because more calories can be administered this way. The use of essential amino acid formulations in feeding can reduce the total amount of protein necessary to meet basic needs. The nutritional management of these disorders is reviewed in depth in the article by (Singh and colleagues) elsewhere in this issue.

Table 1
Emergency pharmacologic dosage in adults

Priming dose	After initial load
Over 90 minutes load with a priming dose of: Sodium phenylacetate and sodium benzoate —5.5 g/m^2 Arginine HCl (10% solution) —200 mg/kg Mix in 10% dextrose intravenous fluid —25–35 ml/kg	Same amounts should be continued on a 24-hour basis

Table 2
Maintenance pharmacologic dosage in adults

CPS1/OTC/NAGS/unknown deficiency	ASS/ASL deficiency
Arginine HCl (10% solution) — 200 mg/kg/24 hrs Sodium phenylacetate and sodium benzoate—5.5 g/m^2/24 hrs	Arginine HCl (10% solution) —600 mg/kg/24 hrs Sodium phenylacetate and sodium benzoate—5.5 g/m^2/24 hrs

Emergency pharmacologic management with ammonia scavengers and arginine is initiated as soon as possible (see dosing recommendations in Table 1), ideally while the dialysis is being arranged and the diagnostic workup is under way. The drug therapy consists of a loading dose followed by a maintenance infusion of sodium phenylacetate/sodium benzoate. Two agents are used in combination to trap nitrogen in excretable forms; they work in molar relationship to the amount of drug given. Because these drugs can be toxic if not administered properly, consultation with an experienced metabolic physician is recommended. Sodium benzoate combines with glycine to make hippurate, which is excreted by the kidneys (or removed in the dialysate), and sodium phenylacetate combines with glutamine to make phenacetylglutamine, which is also excreted in the urine [34,35]. The body replaces these amino acids using excess nitrogen. It is thought that the removal of glutamine by phenylacetate has the additional benefit of removing a compound suspected of having a major role in the neurotoxicity of these disorders [36–41]. Arginine is also used in the acute phase of treatment of urea cycle disorders. In addition to replenishing circulating amino acid levels, arginine can use those parts of the cycle not affected by genetic blocks and incorporate some nitrogen. Because arginine is the precursor for nitric oxide production, it is worth considering modification of the arginine dose downward if the patient develops vasodilation and hypotension.

After the initial loading phase and dialysis, patients should be converted to maintenance doses of the ammonia scavengers (Table 2). If the exact enzyme defect

is known, the amount of arginine administered can be adjusted upward. Of note, in childhood presentations of urea cycle defects, the recommended initial dose of arginine is 600 mg/kg. The effects of this large dose in partial-defect adults has not been extensively studied, and the authors have chosen to recommend the lower 200 mg/kg dose for loading a patient without a diagnosis. If chronic therapy is warranted after the initial treatment, the patient can then be switched to the oral pro-drug of phenylacetate, phenylbutyrate.

Other treatment issues

The use of osmotic agents such as mannitol is not felt to be effective in treating the cerebral edema of hyperammonemia, but this is mainly anecdotal. In canines, opening the blood brain barrier with mannitol resulted in cerebral edema by promoting the entry of ammonia into the brain fluid compartment [42,43]. Two of the patients presented here were placed on mannitol without significant improvement. Intravenous steroids and valproic acid should be avoided. Measures to reduce cerebral metabolism to protect the brain, such as head cooling, have been proposed, but their efficacy is untested. Antibiotics and a septic work-up are indicated to treat potential triggering events. Other measures include physiologic support (pressors, buffering agents to maintain pH, and buffer arginine HCl, and the like) and maintenance of renal output, particularly if ammonia scavengers are being used. Finally, it is imperative to reassess continuation of care after the initial phase of treatment.

Rapid response to the hyperammonemia improves outcome. Symptomatology centers around cerebral edema and pressure on the brain stem. The resulting decrease in cerebral blood flow plus prolonged seizures, when they occur, are poor prognostic factors, as illustrated by our two patients who had protracted seizures and who did not survive. In adults, because the sutures of the skull are fused, sensitivity to hyperammonemia appears considerably greater than in children. Thus treatment should be aggressive and instituted at a lower ammonia concentration than used in children.

Neurologic and treatment evaluation

Cerebral studies should be conducted to determine the efficacy of treatment and whether continuation is warranted. EEG should be performed, because so many of these patients develop status epilepticus. If available, MRI-determined cerebral blood flow can be used to establish if venous stasis has occurred secondary to the cerebral edema. Evaluation of brain stem function and higher cortical function are useful to assess outcome. Finally, the decision for continuation is based on baseline neurologic status, duration of the patient's coma and potential for recovery, and whether the patient is a candidate for transplantation. If the basic urea cycle defect is severe enough, liver transplantation should be considered. Criteria for transplantation are of course linked back to neurologic status, duration of coma, and availability of livers. Diagnostic samples of DNA, liver, and skin should be obtained, because they can be central in family counseling and future treatment issues.

Long-term management

Every effort should be made to avoid similar triggering events. In particular, intravenous steroids for asthma or valproic acid are contraindicated. Long-term diet modification with nutritional oversight is often necessary in patients who have chronic episodes of hyperammonemia. Patients should also avoid dehydration, an especially common occurrence among adults in connection with alcohol intake, hiking, and airline flights. Not all adult patients who recover from a hyperammonemic episode require chronic nitrogen scavengers, but they ought to be considered because many of these patients can become more brittle as time goes on. Special precautions must be taken to avoid catabolism during subsequent illnesses or surgeries, as well as during any event resulting in significant bleeding or tissue damage.

Should psychiatric problems occur over the long term, caregivers should be alert to the possibility of hyperammonemia. Two of the patients discussed had a history of psychiatric illness, suggesting something more than coincidence. In addition, many patients who have citrullinemia type 2, in particular, have presented with mental disturbance [44,45].

It is important to provide genetic counseling to assess risk to other family members.

Summary

Adult patients who have UCDs are being found with increasing frequency. This is most likely due to an increased awareness of these disorders as underlying factors in the intensive care setting. Nevertheless, results can be poor in these patients, even with recognition. The severity of the presentation before diagnosis and the complexities of concomitant clinical factors may explain this. Awareness that the underlying defect has been unmasked by a stressful medical event contributes to understanding the pathophysiology. Moreover, prompt recognition and treatment are key to improving outcomes. Diagnosis is also important to prevent recurrence in the future and to provide counseling for other family members. Clinicians should consider partial inborn errors of metabolism in those patients in the acute care setting whose degree of illness does not fit the apparent clinical causes.

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Exhibit Q

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WEST	470623	268-059-0066-0	9/25/2015 7:10 AM	9/25/2015	10/1/2015 12:07 PM

First Name	Middle Initial	Phone	Control Number	Account Number	Account Phone Number
KENDRIA				09134075	954-766-8433

Date of Birth	Age	Sex	Fasting	Physician Name	Physician ID
[redacted]	48	F	Yes	Hancock S	1245236009

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Tests Ordered

Amino Acid Profile, Qn, Plasma; Ceruloplasmin

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Tests	Result	Flag	Units	Reference Interval	Lab
Amino Acid Profile, Qn, Plasma					
Taurine	51.3		umol/L	29.2-132.3	BN
Aspartate	1.9		umol/L	0.9-7.4	BN
Hydroxyproline	31.4		umol/L	4.7-35.2	BN
Threonine	138.4		umol/L	67.8-211.6	BN
Serine	116.8		umol/L	48.7-145.2	BN
Asparagine	65.9		umol/L	29.5-84.5	BN
Glutamate	43.5		umol/L	18.1-155.9	BN
Glutamine	681.5		umol/L	332.0-754.0	BN
Sarcosine	1.9		umol/L	0.0-4.0	BN
Alpha-aminoadipate	0.6		umol/L	0.0-2.2	BN
Proline	145.3		umol/L	84.8-352.5	BN
Glycine	222.3		umol/L	132.0-467.0	BN
Alanine	332.3		umol/L	124.8-564.2	BN
Citrulline	30.7		umol/L	13.7-63.2	BN
Alpha-aminobutyrate	**36.2**	**High**	umol/L	5.4-34.5	BN
Valine	229.3		umol/L	102.6-345.4	BN
Cystine	35.7		umol/L	13.5-60.2	BN
Methionine	24.8		umol/L	12.7-41.1	BN
Homocitrulline	0.6		umol/L	0.0-1.7	BN
Cystathionine	<0.1		umol/L	0.0-0.7	BN

083

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Lab ID: 470623

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BLOOD TEST

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1-800-208-3444 • LifeExtension.com

WEST, KENDRIA - ID#: 470623

Tests	Result	Flag	Units	Reference Interval	Lab
Amino Acid Profile, Qn, Plasma					
Alloisoleucine	2.2		umol/L	0.4-3.2	BN
Isoleucine	56.8		umol/L	27.7-112.8	BN
Leucine	111.6		umol/L	54.9-205.0	BN
Tyrosine	53.7		umol/L	31.1-118.1	BN
Phenylalanine	54.8		umol/L	33.6-101.9	BN
Argininosuccinate	0.1		umol/L	0.0-3.0	BN
Beta-alanine	2.7		umol/L	1.1-9.0	BN
Beta-aminoisobutyrate	0.7		umol/L	0.3-4.3	BN
Homocystine	<0.1		umol/L	0.0-0.1	BN
Gamma-aminobutyrate	<0.4		umol/L	0.0-0.3	BN
Tryptophan	51.2		umol/L	23.5-93.0	BN
Hydroxylysine	0.2		umol/L	0.1-0.8	BN
Ornithine	53.1		umol/L	30.5-131.4	BN
Lysine	182.7		umol/L	94.0-278.0	BN
Histidine	86.4		umol/L	47.2-98.5	BN
Arginine	68.5		umol/L	32.0-150.0	BN
Interpretation	Comment				TG
	No evidence of aminoacidopathy by quantitative plasma amino acid analysis.				
Director Review	Comment				TG
	Adviye Ayper Tolun, MS, PhD, FACMG Director, Biochemical Genetics To discuss these results or other testing for inborn errors of metabolism, please contact our Biochemical Geneticists at 1-800-345-GENE(4363), LabCorp Genetics Customer Service, RTP, NC.				
Methodology	Comment				TG
	Amino acid concentrations were obtained by LC-MS/MS analysis.				
Ceruloplasmin					
Ceruloplasmin	22.7		mg/dL	16.0-45.0	MB

FINAL REPORT

10/2/2015 12:32:36 PM

LIFE EXTENSION / NATIONAL DIAGNOSTICS, INC

Exhibit Q

LifeExtension® National Diagnostics, Inc. | BLOOD TEST

5990 North Federal Highway, Ft. Lauderdale, FL 33308
1-800-208-3444 • LifeExtension.com

Lab	Facility	Director	Phone
BN	**LabCorp B** 1447 York Court, Burlington, NC,	F, F	800-762-4344
TG	**LabCorp R** 1912 TW Alexander Drive, RTP, NC,	Chatterjee, Chatterjee	800-735-4087
MB	**LabCorp B** 1801 First Avenue South, Birmingham, AL,	Elgin, Elgin	205-581-3500

For inquiries, the physician may contact the above locations.

Thank you for ordering your lab tests through Life Extension/National Diagnostics, Inc. If you would like to discuss your results please call us at 1-800-208-3444. In order to ensure your privacy we ask that you have a copy of your results in front of you when making the call, as you will be asked to provide a specimen number or other identifier from the report. Our advisory team WILL NOT be able to review your lab results with you, unless you are able to provide this information from the report. We also understand that there are times when you will want to review a family members blood test results with our staff. Although Life Extension is happy to comply with these requests, permission (either verbally or in writing) must be given by the person who took the blood tests in order for us to do so. Thank you for your cooperation with these policies as we endeavor to keep your blood test results secure.

FINAL REPORT

10/2/2015 12:32:36 PM

LIFE EXTENSION / NATIONAL DIAGNOSTICS, INC

Exhibit R

LabCorp
Laboratory Corporation of America

Patient Report

Specimen ID: 268-059-0077-0
Control ID: 22725631

Acct #: 17452095 **Phone:** (800) 539-6119 **Rte:** 00
Walk-In Lab, LLC
VART verified
169 W Augusta Lane
SLIDELL LA 70458

WEST, KENDRIA
1109 BLUEWILLOW CT
ANTIOCH TN 37013
(615) 641-1919

Patient Details
DOB: [redacted]
Age(y/m/d): 047/11/20
Gender: F **SSN:**
Patient ID: 22725631

Specimen Details
Date collected: 09/25/2015 0712 Local
Date entered: 09/25/2015
Date reported: 09/29/2015 0849 ET

Physician Details
Ordering: S HANCOCK
Referring:
ID: 1245236009
NPI: 1245236009

General Comments & Additional Information
Alternate Control Number: 22725631
Total Volume: Not Provided

Alternate Patient ID: Not Provided
Fasting: Yes

Ordered Items
Ammonia, Plasma; Venipuncture

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
Ammonia, Plasma	48		ug/dL	19 - 87	01

01 MB LabCorp Birmingham
1801 First Avenue South, Birmingham, AL 35233-1935
John Elgin, MD

For inquiries, the physician may contact **Branch: 504-828-2666 Lab: 205-581-3500**

Date Issued: 09/29/15 1024 ET

FINAL REPORT

Exhibit S

LabCorp
Laboratory Corporation of America

LabCorp Birmingham
1801 First Avenue South
Birmingham, AL 35233-1935
Phone: **205-581-3500**

Specimen Number	Patient ID	Control Number	Account Number	Account Phone Number	Route
268-059-0060-0	267059	EC1527849	17123375	985-624-9186	40

Patient Last Name	Patient First Name	Patient Middle Name	Patient SS#	Patient Phone	Total Volume	Age (Y/M/D)	Sex	Fasting
WEST	KENDRIA	Y		615-641-1919		47/11/20	F	Yes

[redacted]dress

Account Address
Direct Laboratory Services LLC
Interface Acct
4040 Florida Street Suite 101
Mandeville LA 70448

Additional Information

Date and Time Collected	Date Entered	Date and Time Reported	Physician Name	NPI	Physician ID
09/25/15 07:08	09/25/15	09/26/15 10:13ET	HAASE , D	1457330771	1457330771

Tests Ordered
CMP12+LP+6AC; CBC With Differential/Platelet; Iron and TIBC; Ferritin, Serum; Transferrin; Venipuncture

General Comments
ACC: EC1527849 PID:

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
CMP12+LP+6AC					
Chemistries					01
Glucose, Serum	87		mg/dL	65 - 99	01
Uric Acid, Serum	4.9		mg/dL	2.5 - 7.1	01
Please Note:					01
	Therapeutic target for gout patients: <6.0				
BUN	12		mg/dL	6 - 24	01
Creatinine, Serum	0.89		mg/dL	0.57 - 1.00	01
eGFR If NonAfricn Am	77		mL/min/1.73	>59	
eGFR If Africn Am	89		mL/min/1.73	>59	
BUN/Creatinine Ratio	13			9 - 23	
Sodium, Serum	140		mmol/L	134 - 144	01
Potassium, Serum	4.3		mmol/L	3.5 - 5.2	01
Chloride, Serum	102		mmol/L	97 - 108	01
Calcium, Serum	8.9		mg/dL	8.7 - 10.2	01
Phosphorus, Serum	3.6		mg/dL	2.5 - 4.5	01
Protein, Total, Serum	7.2		g/dL	6.0 - 8.5	01
Albumin, Serum	4.2		g/dL	3.5 - 5.5	01
Globulin, Total	3.0		g/dL	1.5 - 4.5	
A/G Ratio	1.4			1.1 - 2.5	
Bilirubin, Total	0.5		mg/dL	0.0 - 1.2	01
Alkaline Phosphatase, S	73		IU/L	39 - 117	01
LDH	134		IU/L	119 - 226	01
AST (SGOT)	24		IU/L	0 - 40	01
ALT (SGPT)	24		IU/L	0 - 32	01
GGT	34		IU/L	0 - 60	01
Iron, Serum	100		ug/dL	35 - 155	01
Lipids					01
Cholesterol, Total	184		mg/dL	100 - 199	01
Triglycerides	58		mg/dL	0 - 149	01
HDL Cholesterol	66		mg/dL	>39	01
Comment					01

LabCorp
Laboratory Corporation of America

LabCorp Birmingham
1801 First Avenue South
Birmingham, AL 35233-1935

Phone: 205-581-3500

Patient Name					Specimen Number		
WEST, KENDRIA Y					268-059-0060-0		
Account Number	Patient ID	Control Number	Date and Time Collected	Date Reported	Sex	Age(Y/M/D)	Date of Birth
17123375	267059	EC1527849	09/25/15 07:08	09/26/15	F	47/11/20	[redacted]

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
According to ATP-III Guidelines, HDL-C >59 mg/dL is considered a negative risk factor for CHD.					
VLDL Cholesterol Cal	12		mg/dL	5 - 40	
LDL Cholesterol Calc	**106**	**High**	mg/dL	0 - 99	
T. Chol/HDL Ratio	2.8		ratio units	0.0 - 4.4	
Please Note:					01
				T. Chol/HDL Ratio Men Women	
				1/2 Avg.Risk 3.4 3.3	
				Avg.Risk 5.0 4.4	
				2X Avg.Risk 9.6 7.1	
				3X Avg.Risk 23.4 11.0	
Estimated CHD Risk	< 0.5		times avg.	0.0 - 1.0	
				T. Chol/HDL Ratio Men Women	
				1/2 Avg.Risk 3.4 3.3	
				Avg.Risk 5.0 4.4	
				2X Avg.Risk 9.6 7.1	
				3X Avg.Risk 23.4 11.0	
The CHD Risk is based on the T. Chol/HDL ratio. Other factors affect CHD Risk such as hypertension, smoking, diabetes, severe obesity, and family history of premature CHD.					
CBC With Differential/Platelet					
WBC	4.9		x10E3/uL	3.4 - 10.8	01
RBC	4.18		x10E6/uL	3.77 - 5.28	01
Hemoglobin	13.3		g/dL	11.1 - 15.9	01
Hematocrit	41.0		%	34.0 - 46.6	01
MCV	**98**	**High**	fL	79 - 97	01
MCH	31.8		pg	26.6 - 33.0	01
MCHC	32.4		g/dL	31.5 - 35.7	01
RDW	14.2		%	12.3 - 15.4	01
Platelets	186		x10E3/uL	150 - 379	01
Neutrophils	48		%		01
Lymphs	40		%		01
Monocytes	9		%		01
Eos	3		%		01
Basos	0		%		01
Neutrophils (Absolute)	2.3		x10E3/uL	1.4 - 7.0	01
Lymphs (Absolute)	2.0		x10E3/uL	0.7 - 3.1	01
Monocytes(Absolute)	0.5		x10E3/uL	0.1 - 0.9	01
Eos (Absolute)	0.2		x10E3/uL	0.0 - 0.4	01
Baso (Absolute)	0.0		x10E3/uL	0.0 - 0.2	01
Immature Granulocytes	0		%		01
Immature Grans (Abs)	0.0		x10E3/uL	0.0 - 0.1	01

Exhibit S

LabCorp
Laboratory Corporation of America

LabCorp Birmingham
1801 First Avenue South
Birmingham, AL 35233-1935
Phone: **205-581-3500**

Patient Name	Specimen Number
WEST, KENDRIA Y	**268-059-0060-0**

Account Number	Patient ID	Control Number	Date and Time Collected	Date Reported	Sex	Age(Y/M/D)	Date of Birth
17123375	267059	EC1527849	09/25/15 07:08	09/26/15	F	47/11/20	[redacted]

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
Iron and TIBC					
Iron Bind.Cap.(TIBC)	305		ug/dL	250 - 450	
UIBC	205		ug/dL	150 - 375	01
Iron Saturation	33		%	15 - 55	
Ferritin, Serum	69		ng/mL	15 - 150	01
Transferrin	277		mg/dL	200 - 370	01

01 MB LabCorp Birmingham Dir: John Elgin, MD
1801 First Avenue South, Birmingham, AL 35233-1935
For inquiries, the physician may contact **Branch: 800-762-4344 Lab: 205-581-3500**

Exhibit T

SpectraCell Laboratories
Science + Health + Solutions

LABORATORY REPORT

Account Number: 274187

Kendria West
10401 Town Park Drive
Houston, TX 77072
USA

Name: **Kendria West**
Gender: Female
[redacted]

Accession Number: P22505
Requisition Number:

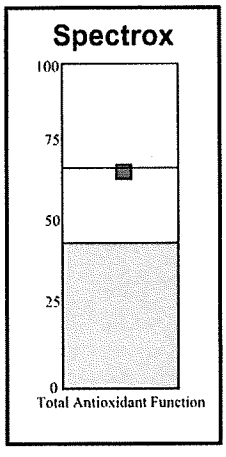
Date of Collection: 09/28/2015
Date Received: 09/29/2015
Date Reported: 10/08/2015

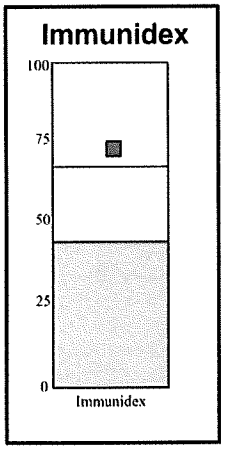
Summary of Deficient Test Results

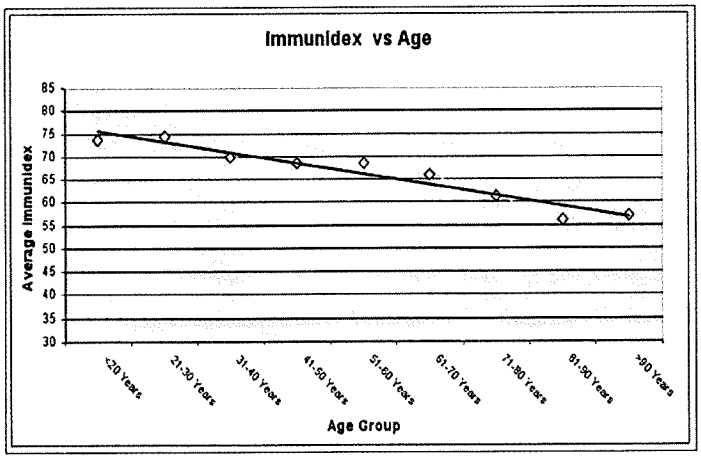
Testing determined the following functional deficiencies:

Vitamin B2 Vitamin D3 Vitamin K2

Supplementation to correct functional micronutrient deficiences without medical approval can be detrimental in some common medical conditions. Consult with your physician before starting repletion.







John F. Crawford, Ph.D.
Laboratory Director

CLIA# 45D0710715

All tests performed by SpectraCell Laboratories, Inc. * 10401 Town Park Drive Houston, TX 77072
Tel (713) 621-3101 * Toll-free (800)-227-LABS(5227) * Fax (713) 621-3234 * www.spectracell.com

OVERVIEW OF TEST PROCEDURE

1. A mixture of lymphocytes is isolated from the blood.
2. These cells are grown in a defined culture medium containing optimal levels of all essential. nutrients necessary to sustain their growth in cell culture.
3. The T-lymphocytes are stimulated to grow with a mitogen (phytohemagglutinin) and growth is measured by the incorporation of tritiated (radioactive) thymidine into the DNA of the cells.

The growth response under optimal conditions is defined as 100%, and all other growth rates are compared to this 100% level of growth.

For example – we remove vitamin B6 from the medium and stimulate the cells to grow by mitogen stimulation. Growth is measured by DNA synthesis and the rate of growth is dependent only upon the functional level of vitamin B6 available within the cells to support growth. For Vitamin B6 a growth rate of at least 55% of the growth rate observed in the optimal (100%) media is considered normal. Results less than 55% are considered to indicate a functional deficiency for Vitamin B6. Each nutrient has a different reference range that was established by assaying thousands of apparently healthy individuals.

BREAKING DOWN THE REPORT

1. TEST RESULT (% CONTROL)

This column represents the patient's growth response in the test media measured by DNA synthesis as compared to the optimal growth observed in the 100% media.

2. FUNCTIONAL ABNORMALS

An interpretation is provided for those nutrients found to be deficient.

3. REFERENCE RANGE

This column represents how this patient's result compares to thousands of patients previously tested. A patient's result is considered deficient when it is less than the reference range.

4. GRAPHS

The abnormal range of results is noted in the blue area. Abnormal results are indicated in red. The gray cross hatch area is a representation of the range of test results found in a random selection of subjects.

SPECTROX® – TOTAL ANTIOXIDANT FUNCTION

SPECTROX® is a measurement of overall antioxidant function. The patient's cells are grown in the optimal media, stimulated to grow, and then increasing amounts of a free radical generating system (H_2O_2) are added. The cell's ability to resist oxidative damage is determined. The increasing levels of peroxide will result in diminished growth rates in those patients with poor antioxidant function capacity.

INDIVIDUAL ANTIOXIDANT LEVELS

In the tests for individual antioxidants, it is determined which specific antioxidants may be deficient and thus affecting the SPECTROX® antioxidant function result. For these tests, the patient's cells are preincubated with one of the nutrient antioxidants, i.e. selenium, and then the Spectrox® test is repeated to determine if the addition of selenium improves the patient's antioxidant function. This process is repeated for each individual antioxidant.

Antioxidants tested with this process:
Glutathione, Cysteine, Coenzyme-Q10, Selenium, Vitamin E, Alpha Lipoic Acid, and Vitamin C.

SpectraCell Laboratories, Inc.
Laboratory Test Report

Accession Number: P22505
Kendria West

Repletion Suggestions

1. Vitamin B2 (Riboflavin)	20 mg daily of Riboflavin or Riboflavin-5-Phosphate
2. Vitamin D3 (Cholecalciferol)	1000 IU daily of Cholecalciferol (Vitamin D3-1-alpha 25-dihydroxyvitamin D)
3. Vitamin K2	100 mcg vitamin K1 (K2 precursor) daily

Please note: Supplementation is usually required for four to six months to effect the repletion of a functional deficiency in lymphocytes

Suggestions for supplementation with specific micronutrients must be evaluated and approved by the attending physician. This decision should be based upon the clinical condition of the patient and the evaluation of the effects of supplementation on current treatment and medication of the patient.

SpectraCell Laboratories, Inc.
Laboratory Test Report

Accession Number: P22505
Kendria West

Micronutrients	Patient Results (% Control)	Functional Abnormals	Reference Range (greater than)
B Complex Vitamins			
Vitamin B1 (Thiamin)	103		>78%
Vitamin B2 (Riboflavin)	53	Deficient	>53%
Vitamin B3 (Niacinamide)	98		>80%
Vitamin B6 (Pyridoxine)	72		>54%
Vitamin B12 (Cobalamin)	16		>14%
Folate	37		>32%
Pantothenate	14		>7%
Biotin	55		>34%
Amino Acids			
Serine	47		>30%
Glutamine	58		>37%
Asparagine	52		>39%
Metabolites			
Choline	31		>20%
Inositol	75		>58%
Carnitine	56		>46%
Fatty Acids			
Oleic Acid	72		>65%
Other Vitamins			
Vitamin D3 (Cholecalciferol)	49	Deficient	>50%
Vitamin A (Retinol)	75		>70%
Vitamin K2	30	Deficient	>30%
Minerals			
Calcium	48		>38%
Manganese	67		>50%
Zinc	44		>37%
Copper	55		>42%
Magnesium	47		>37%
Carbohydrate Metabolism			
Glucose-Insulin Interaction	53		>38%
Fructose Sensitivity	51		>34%
Chromium	44		>40%
Antioxidants			
Glutathione	57		>42%
Cysteine	53		>41%
Coenzyme Q-10	94		>86%
Selenium	82		>74%
Vitamin E (A-tocopherol)	91		>84%
Alpha Lipoic Acid	87		>81%
Vitamin C	56		>40%
SPECTROX™			
Total Antioxidant Function	66		>40%
Proliferation Index			
Immunidex	70		>40%

The reference ranges listed in the above table are valid for male and female patients 12 years of age or older.

Exhibit T

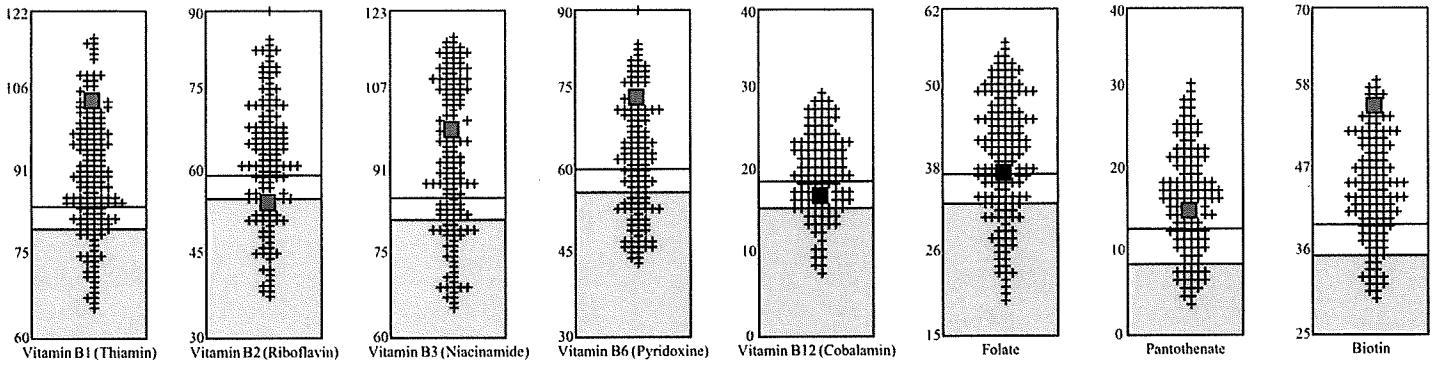
Adequate
Borderline
Deficient

Deficient: Values in this area represent a deficiency and may require nutrient repletion or dietary changes

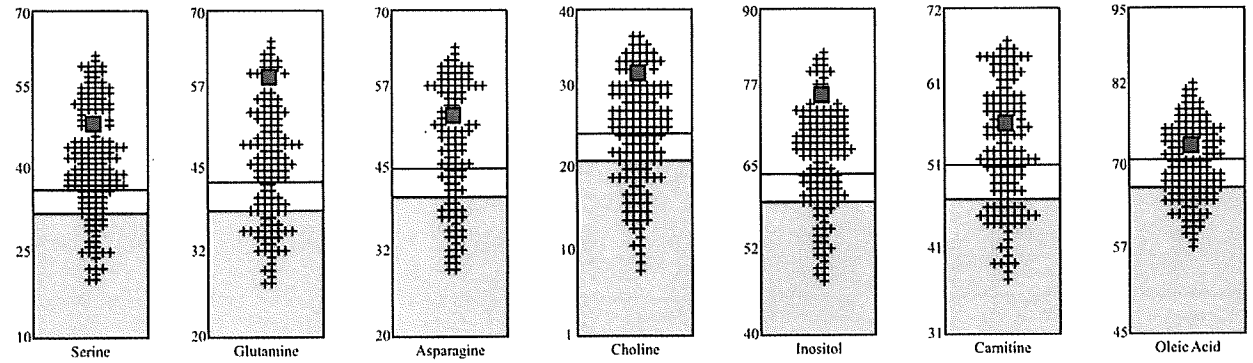
Borderline: Values in this area represent a borderline and may require nutrient repletion or dietary changes.

Accession Number: P22505
Kendria West

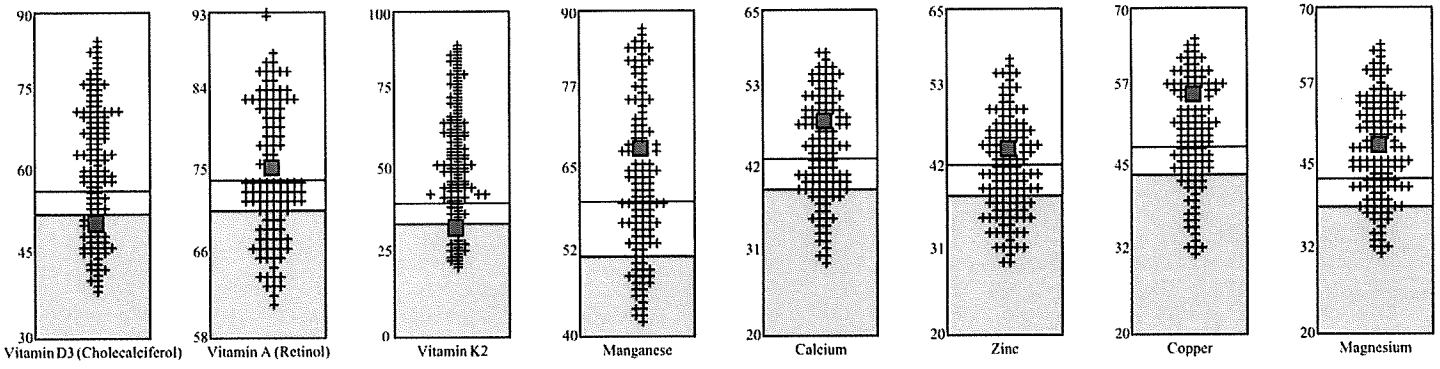
B Complex Vitamins



Amino Acids & Metabolites



Other Vitamins & Minerals



Adequate
Borderline
Deficient

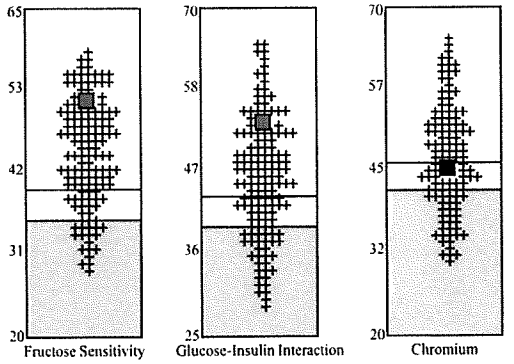
Deficient: Values in this area represent a deficiency and may require nutrient repletion or dietary changes

Borderline: Values in this area represent a borderline and may require nutrient repletion or dietary changes.

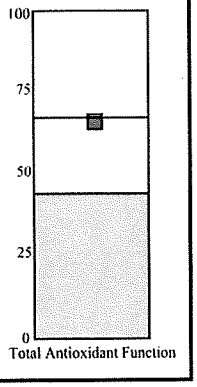
Exhibit T

Accession Number: P22505
Kendria West

Carbohydrate Metabolism



Spectrox

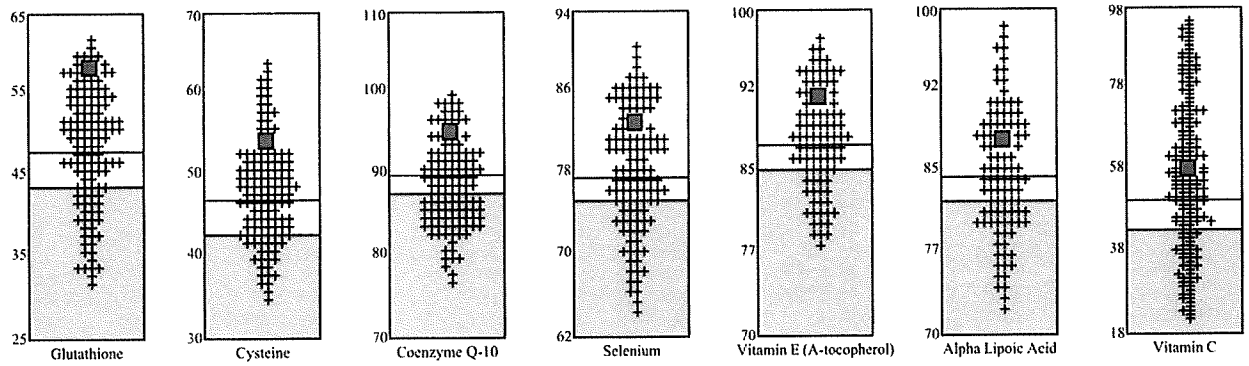


A Spectrox value above 65%-
indicates a desirable status for apparently healthy individuals. Since antioxidants are protective nutrients, the most desired status would be the greatest ability to resist oxidative stress.

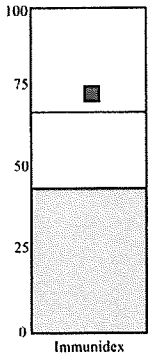
A Spectrox value between 40% and 65%-
indicates an average antioxidant function for apparently healthy individuals. An average status means the ability to resist oxidative stress similar to the majority of persons. However, average status is not ideal, nor is it clearly deficient.

A Spectrox value below 40%-
indicates a deficient antioxidant function resulting in a decreased ability to resist oxidative stress or an increased antioxidant load.

Individual Antioxidants



Immunidex



The Immunidex is an indication of the patient's T-Lymphoproliferative response to mitogen stimulation relative to the response of a control population. An average or weakened immune response may improve with correction of the nutritional deficiencies determined by the micronutrient testing.

An Immunidex above 65%- indicates a strong response, a measurement of cell-mediated immune function.

An Immunidex between 40% and 65% - indicates an average response.

An Immunidex below 40%- may indicate a weakened cell mediated immune response.

SpectraCell Laboratories
Science + Health + Solutions

Comprehensive Thyroid Plus Adrenal Report

Component Summaries

This information is provided for educational purposes.

TSH (Thyroid Stimulating Hormone)

This hormone (also known as thyrotropin) tells the thyroid to increase or decrease production of T4 or conversion to T3 depending on the amounts of T4 and T3 circulating in the bloodstream via an efficient feedback system. Levels of TSH are high when thyroid function is poor or inefficient (hypothyroidism) because TSH is released by a gland in the brain (pituitary) in an effort to increase thyroid function by increasing T4 or T3. Conversely, low TSH is seen in an overactive thyroid (hyperthyroidism). TSH is often considered the main thyroid hormone for diagnosing hypo- or hyperthyroidism.

T4, Total (Total Thyroxine)

Most T4 in the blood is bound to carrier proteins which makes it biologically inactive. Total T4 includes unbound (free) T4 plus T4 that is bound to carrier proteins in the blood.

T4, Free (Free Thyroxine)

Considered a precursor hormone, T4 is converted into T3 as required by cells throughout the body. Generally, this conversion of T4 to T3 occurs outside the thyroid gland, typically in the liver and kidneys. Although T4 is more abundant in the blood than T3, it is much less potent.

T3, Free (Free Triiodothyronine)

T3 is the main thyroid hormone in terms of biological activity that regulates metabolism and growth throughout the body. It is more potent than T4 and directly affects the heart, blood vessels, bone, muscle and brain. T3 increases a person's metabolic rate, controls body temperature, regulates neurotransmitter synthesis (mood), impacts heart rate and oversees the conversion of food into energy.

Tg (Thyroglobulin)

The main function of Tg is to store iodine, which is a necessary nutrient for the production of thyroid hormones T3 and T4. This test is particularly useful when monitored over time versus a single measurement and can sometimes be a useful tumor marker in patients with previous thyroid cancer.

TBG (Thyroid Binding Globulin)

TBG is a carrier protein for thyroid hormones so its role is to transport T4 and T3 through the bloodstream. The thyroid gland adjusts to changing levels of TBG in order to keep free T4 constant and it is particularly useful when thyroid (T4) levels do not necessarily correlate with clinical symptoms. TBG levels are largely affected by other hormones and many prescription drugs and is useful in diagnosing the reason behind abnormal thyroid hormone levels.

Anti-Tg (Antibodies to Thyroglobulin)

If antibodies to the protein thyroglobulin (a precursor to T4) are present in significant amounts, this suggests an abnormal immune response against your own body, also called autoimmunity. Specifically, anti-Tg suggests a person's immune system is attacking healthy tissue – in this case, the protein precursor to thyroid hormone.

Anti-TPO (Antibodies to Thyroperoxidase)

Thyroperoxidase (TPO) is an enzyme that initiates the synthesis of T4. Antibodies to TPO indicate autoimmunity where the body is attacking normal proteins in the blood (in this case, TPO). People with anti-TPO have a higher chance of developing hypothyroidism that those who do not have antibodies to TPO.

DHEA-S

Dehydroepiandrosterone sulfate (DHEA-S) – the most abundant sex hormone in the body, DHEA-S (the sulfated, or bioavailable form of DHEA), is produced by the adrenal glands and is the precursor hormone to testosterone and estrogens. DHEA-S enhances immunity, reduces autoimmunity, helps prevent cancer, and improves insulin sensitivity, bone health and cognitive function.

Cortisol

This steroid hormone is secreted by the adrenal glands in response to physical or psychological stress. The short-term effects of cortisol help muscles use glucose for immediate energy but prolonged cortisol secretion negatively alters blood sugar and fat metabolism and may predispose a person toward insulin resistance. Cortisol supresses the immune system, increases blood pressure, reduces bone formation and inhibits anabolic (tissue building) functions such as collagen synthesis. Significant fluctuations in cortisol levels occur throughout the day peaking in early morning and dipping late at night in healthy individuals.

Reverse T3 (Reverse Triiodothyronine)

As the name implies, Reverse T3 opposes the biological action of T3. It slows metabolism and renders T3 in the body biologically inactive. The rate of rT3 production relative to T3 will increase in times of stress (high cortisol) and in the presence of nutrient deficiencies, inflammation or certain medications.

Exhibit U

SpectraCell Laboratories
Science + Health + Solutions

Comprehensive Thyroid Plus Adrenal Report

Patient Name:	West, Kendria	Batch Number:	B6707
Patient DOB:	[redacted]	Accession Number:	P45417
Gender:	F	Date Received:	12/19/2015
Physician	Kendria West	Report Date:	12/28/2015

Test Component		Flag	Result	Reference Range
DHEA-S	µg/dL		80	35 - 430
TSH	µIU/mL		1.760	0.358 - 3.74
T4, Total	µg/dL		7.1	4.5 - 12.5
T3, Free	pg/mL		3.9	2.2 - 4.0
T4, Free	ng/dL		1.05	0.76 - 1.46
Cortisol	µg/dL		11.5	3.1 - 22.4
Anti-TPO Ab	IU/mL		< 10.0	0.0 - 35.0
Anti-Thyroglobulin Ab	IU/mL		< 20	ND - 40
Thyroglobulin	ng/mL		11	<= 55
Thyroxine-binding globulin, TBG	µg/mL		18	14 - 31

SpectraCell Laboratories
Science + Health + Solutions

Exhibit U

Comprehensive Thyroid Plus Adrenal Report

Patient Name: West, Kendria
Patient DOB: [redacted]
Gender: F
Physician Kendria West

Batch Number: B6707
Accession Number: P45417
Date Received: 12/19/2015
Report Date: 12/28/2015

Test			Patient Results	Reference Value
DHEA-S	µg/dL	0 125 250 375 500	80	35 - 430
TSH	µIU/mL	0.020 1.260 2.500 3.740 4.980	1.760	0.358 - 3.74
T4, Total	µg/dL	0.5 4.4 8.3 12.1 16.0	7.1	4.5 - 12.5
T3, Free	pg/mL	0.5 1.9 3.3 4.6 6.0	3.9	2.2 - 4.0
T4, Free	ng/dL	0.10 0.83 1.55 2.28 3.00	1.05	0.76 - 1.46
Cortisol	µg/dL	0.2 11.4 22.6 33.8 45.0	11.5	3.1 - 22.4
Anti-TPO Ab	IU/mL	+ 10.0 20.0 30.0 40.0 50.0	< 10.0	0.0 - 35.0
Anti-Thyroglobulin Ab	IU/mL	+ 20 30 40 50 60	< 20	ND - 40
Thyroglobulin	ng/mL	0 20 40 60 80	11	<= 55
Thyroxine-binding globulin, TBG	µg/mL	0 11 23 34 45	18	14 - 31

SpectraCell Laboratories
Science + Health + Solutions

Hormone Balance - Female Report

Component Summaries

This information is provided for educational purposes.

Estradiol (E2)
The strongest estrogen, E2 protects blood vessels, increases high density lipoprotein cholesterol (HDL), prevents bone loss, helps form collagen which benefits the appearance of the skin, improves cognitive function and increases the immune response. However, estradiol also exerts a strong proliferative effect on hormone sensitive tissues like the breast, uterus and ovary so it must be properly balanced with progesterone and other estrogens to prevent the clinical manifestation of estrogen dominance.

Progesterone
Progesterone selectively balances the effects of estrogen in hormonally sensitive tissue (breast, uterine) as well as in the bones, brain, and skin. It decreases the immune response, promotes bone formation, protects the brain and tends to have a calming effect on mood. It is also a precursor hormone for other sex hormones as well as cortisol and interacts with thyroid hormones to regulate metabolism.

FSH (Follicle Stimulating Hormone)
FSH stimulates the production of estrogens and is a marker for ovarian function in women. Levels of FSH increase during both ovulation and during ovarian failure and is considered an appropriate test for determining menopausal status in women.

LH (Luteinizing Hormone)
LH is responsible for ovulation in premenopausal women and works synergistically with follicle stimulating hormone to ensure female fertility. LH surges mid-menstrual cycle in women and initiates the release of progesterone. It regulates estrogen production in the ovary and is largely affected by prolactin levels.

Prolactin
Prolactin is an inhibitory hormone that reduces the action of several other hormones. Most known for its ability to stimulate milk production in lactating women, it also regulates calcium metabolism and plays a role in the synthesis of nerve cells and prostaglandins, which are hormone-like substances that regulate inflammation and metabolic processes throughout the body.

IGF-1 (Insulin-like Growth Factor 1)
IGF-1 is an anabolic (tissue building) hormone that is similar in structure (not function) to insulin. Working intimately with growth hormone, IGF-1 causes cells to grow in several tissues throughout the body including muscle, bone, nerves, skin and various organs.

DHEA-S (Dehydroepiandrosterone sulfate)
The most abundant sex hormone in the body, DHEA-S is produced primarily in adrenal glands and is the main precursor hormone for androgens (estrogen & testosterone). DHEA-S enhances immunity, decreases autoimmunity, helps prevent cancer, and improves insulin sensitivity, cognitive function and bone health.

Testosterone
Although levels in women are 5-10% the amount found in men, testosterone is a potent steroid hormone that is clinically associated with increased muscle mass, libido, bone health and a general sense of well being in women. It can also be converted to estrogens and is regulated by LH and FSH. Only free, unbound testosterone is biologically active. Testosterone that is bound to SHBG is basically inert so free testosterone can be calculated if the amount of SHBG in the blood is also known.

Androstenedione
Androstenedione is made from DHEAS and is the immediate precursor hormone to testosterone and estrogen. (DHEAS → Androstenedione → Testosterone → Estrogen). Androstenedione occurs in equilibrium with testosterone so an increase in one usually increases the other.

SHBG (Sex Hormone Binding Globulin)
SHBG, which is regulated by other hormones, is a protein that binds estrogens and testosterone in the bloodstream where they are biologically inactive. Assists in regulation of estrogen and testosterone levels.

Estrone (E1)
This estrogen has very strong tissue proliferative effects and may be linked to estrogen dominant conditions like fibrocystic breasts, endometriosis and uterine fibroids. It will create either dangerous or beneficial metabolites, depending on a person's nutritional status.

Estriol, unconjugated (UE3)
Estriol is a weak estrogen that is very high during pregnancy, but also plays an important role in non-pregnant women by opposing the growth of cancer cells promoted by the stronger estrogens E1 and E2. Estriol is also known to alleviate menopausal symptoms such as hot flashes or urinary incontinence.

SpectraCell Laboratories
Science + Health + Solutions

Exhibit V

Hormone Balance Report

Patient Name: West, Kendria
Patient DOB: [redacted]
Gender: F
Physician Kendria West

Batch Number: B6707
Accession Number: P45417
Date Received: 12/19/2015
Report Date: 12/28/2015

Test Component		Flag	Result	Reference Range	Phase
Estrone (E1)	pg/mL		19.6	< 150	Follicular Phase - Early
Estradiol (E2)	pg/mL		60	ND - 160	Follicular Phase
Estriol, Unconjugated (UE3)	ng/mL		< 0.07	< 0.08	Nonpregnant
Luteinizing Hormone	mIU/mL		5.4	1.1 - 11.6	Follicular Phase
Follicle Stimulating Hormone	mIU/mL	H	14.2	2.8 - 11.3	Follicular Phase
Progesterone	ng/mL	L	0.3	0.33 - 1.2	Follicular Phase
Prolactin	ng/mL		14.3	1.9 - 23.1	
Testosterone, Total	ng/dL		< 20	ND - 73	Ovulating
Testosterone, Free (Calculation)	ng/dL	L	< 0.3	0.3 - 1.9	
SHBG	nmol/L		56	18 - 144	Nonpregnant
Androstenedione	ng/mL		0.8	0.3 - 3.3	
DHEA-S	µg/dL		80	35 - 430	
IGF-1	ng/mL		180	94 - 252	

12/16/2015
Estrone (E1) performed at Sonic Reference Laboratory, Inc. 9200 Wall St., Suite 200, Austin, TX 78754 CLIA# 45D2083658

SpectraCell Laboratories
Science + Health + Solutions

Exhibit V

Hormone Balance Report

Patient Name:	West, Kendria	Batch Number:	B6707
Patient DOB:	[redacted]	Accession Number:	P45417
Gender:	F	Date Received:	12/19/2015
Physician:	Kendria West	Report Date:	12/28/2015

Test		Graph	Patient Results	Reference Range	
Estrone (E1)	pg/mL	0.0 50.0 100.0 150.0 200.0	19.6	< 150	Follicular Phase - Early
Estradiol (E2)	pg/mL	0 62 124 186 248	60	ND - 160	Follicular Phase
Estriol, Unconjugated (UE3)	ng/mL	0.00 0.04 0.08 0.12 0.16	< 0.07	< 0.08	Nonpregnant
Luteinizing Hormone	mIU/mL	0.0 5.0 10.0 15.0 20.0	5.4	1.1 - 11.6	Follicular Phase
Follicle Stimulating Hormone	mIU/mL	0.0 2.8 5.7 8.5 11.3+	14.2	2.8 - 11.3	Follicular Phase
Progesterone	ng/mL	0.0 0.5 1.0 1.5 2.0	0.3	0.33 - 1.2	Follicular Phase
Prolactin	ng/mL	0.0 6.3 12.5 18.8 25.0	14.3	1.9 - 23.1	
Testosterone, Total	ng/dL	0 23 45 68 90	< 20	ND - 73	Ovulating
Testosterone, Free (Calculation)	ng/dL	0.0 1.0 2.0 3.0 4.0	< 0.3	0.3 - 1.9	
SHBG	nmol/L	2 45 89 132 175	56	18 - 144	Nonpregnant
Androstenedione	ng/mL	0.0 1.3 2.5 3.8 5.0	0.8	0.3 - 3.3	
DHEA-S	µg/dL	0 125 250 375 500	80	35 - 430	
IGF-1	ng/mL	0 75 150 225 300	180	94 - 252	

12/16/2015
Estrone (E1) performed at Sonic Reference Laboratory, Inc. 9200 Wall St., Suite 200, Austin, TX 78754 CLIA# 45D2083658

Exhibit W

Amino Acids (24hr Urine)

Patient Copy

GENOVA DIAGNOSTICS

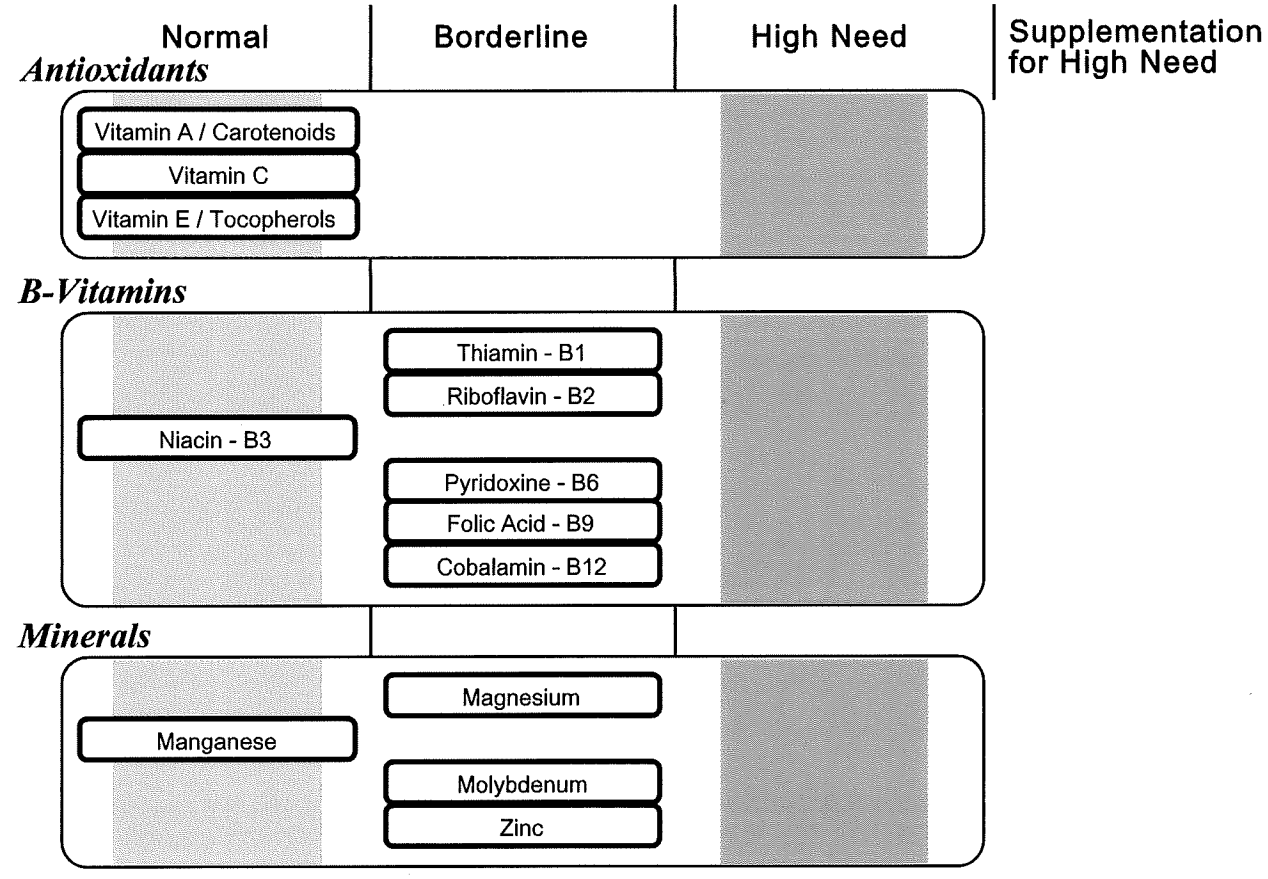
63 Zillicoa Street
Asheville, NC 28801

Patient: KENDRIA WEST
DOB: [redacted]
Sex: F
MRN: 1232614531

Order Number: J3120727
Completed: January 15, 2016
Received: January 12, 2016
Collected: January 11, 2016

True Health: Center for Functional Medicine
Brady Hurst DC
3300 Windyridge Parkway
Ste 403
Atlanta, GA 30339

Results Overview



SUGGESTED SUPPLEMENT SCHEDULE

Supplements	Daily Recommended Intake (DRI)	Patient's Daily Recommendations	Provider Daily Recommendations
Antioxidants			
Vitamin A / Carotenoids	2,333 IU	3,000 IU	
Vitamin C	75 mg	250 mg	
Vitamin E / Tocopherols	22 IU	100 IU	
B-Vitamins			
Thiamin - B1	1.1 mg	25 mg	
Riboflavin - B2	1.1 mg	25 mg	
Niacin - B3	14 mg	20 mg	
Pyridoxine - B6	1.3 mg	25 mg	
Folic Acid - B9	400 mcg	800 mcg	
Cobalamin - B12	2.4 mcg	500 mcg	
Minerals			
Magnesium	320 mg	600 mg	
Manganese	1.8 mg	3.0 mg	
Molybdenum	45 mcg	150 mcg	
Zinc	8 mg	20 mg	
Digestive Support			
Pancreatic Enzymes		5,000 IU	

Amino Acid	mg/day	Amino Acid	mg/day
Arginine	0	Methionine	0
Asparagine	0	Phenylalanine	0
Cysteine	0	Serine	0
Glutamine	0	Taurine	0
Glycine	0	Threonine	0
Histidine	0	Tryptophan	0
Isoleucine	0	Tyrosine	0
Leucine	0	Valine	0
Lysine	231		

Recommendations for age and gender-specific supplementation are set by comparing levels of nutrient functional need to optimal levels as described in the peer-reviewed literature. They are provided as guidance for short-term support of nutritional deficiencies only.

The Suggested Supplemental Schedule is provided at the request of the ordering practitioner. Any application of it as a therapeutic intervention is to be determined by the ordering practitioner.

Key: Normal | Borderline | High Need

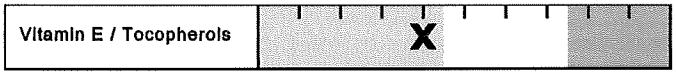
Amino Acids, 24hr Urine Interpretation At-A-Glance

Nutritional Needs

Antioxidants

- Beta-carotene & other carotenoids are converted to vitamin A (retinol), involved in vision, antioxidant & immune function, gene expression & cell growth.
- Vitamin A deficiency may occur with chronic alcoholism, zinc deficiency, hypothyroidism, or oral contraceptives containing estrogen & progestin.
- Deficiency may result in night blindness, impaired immunity, healing & tissue regeneration, increased risk of infection, leukoplakia or keratosis.
- Food sources include cod liver oil, fortified cereals & milk, eggs, sweet potato, pumpkin, carrot, cantaloupe, mango, spinach, broccoli, kale & butternut squash.

- Vitamin C is an antioxidant (also used in the regeneration of other antioxidants). It is involved in cholesterol metabolism, the production & function of WBCs and antibodies, and the synthesis of collagen, norepinephrine and carnitine.
- Deficiency may occur with oral contraceptives, aspirin, diuretics or NSAIDs.
- Deficiency can result in scurvy, swollen gingiva, periodontal destruction, loose teeth, sore mouth, soft tissue ulcerations, or increased risk of infection.
- Food sources include oranges, grapefruit, strawberries, tomato, sweet red pepper, broccoli and potato.



- Alpha-tocopherol (body's main form of vitamin E) functions as an antioxidant, regulates cell signaling, influences immune function and inhibits coagulation.
- Deficiency may occur with malabsorption, cholestyramine, colestipol, isoniazid, orlistat, olestra and certain anti-convulsants (e.g., phenobarbital, phenytoin).
- Deficiency may result in peripheral neuropathy, ataxia, muscle weakness, retinopathy, and increased risk of CVD, prostate cancer and cataracts.
- Food sources include oils (olive, soy, corn, canola, safflower, sunflower), eggs, nuts, seeds, spinach, carrots, avocado, dark leafy greens and wheat germ.

Key

- Function
- Causes of Deficiency
- Complications of Deficiency
- Food Sources

Amino Acids, 24hr Urine Interpretation At-A-Glance

Nutritional Needs

B-Vitamins

Thiamin - B1

- B1 is a required cofactor for enzymes involved in energy production from food, and for the synthesis of ATP, GTP, DNA, RNA and NADPH.
- Low B1 can result from chronic alcoholism, diuretics, digoxin, oral contraceptives and HRT, or large amounts of tea & coffee (contain anti-B1 factors).
- B1 deficiency may lead to dry beriberi (e.g., neuropathy, muscle weakness), wet beriberi (e.g., cardiac problems, edema), encephalopathy or dementia.
- Food sources include lentils, whole grains, wheat germ, Brazil nuts, peas, organ meats, brewer's yeast, blackstrap molasses, spinach, milk & eggs.

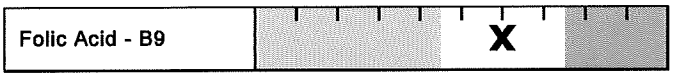
Pyridoxine - B6

- B6 (as P5P) is a cofactor for enzymes involved in glycogenolysis & gluconeogenesis, and synthesis of neurotransmitters, heme, B3, RBCs and nucleic acids.
- Low B6 may result from chronic alcoholism, long-term diuretics, estrogens (oral contraceptives and HRT), anti-TB meds, penicillamine, L-DOPA or digoxin.
- B6 deficiency may result in neurologic symptoms (e.g., irritability, depression, seizures), oral inflammation, impaired immunity or increased homocysteine.
- Food sources include poultry, beef, beef liver, fish, whole grains, wheat germ, soybean, lentils, nuts & seeds, potato, spinach and carrots.

Riboflavin - B2

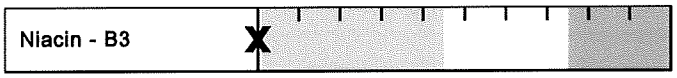
- B2 is a key component of enzymes involved in antioxidant function, energy production, detoxification, methionine metabolism and vitamin activation.
- Low B2 may result from chronic alcoholism, some anti-psychotic medications, oral contraceptives, tricyclic antidepressants, quinacrine or adriamycin.
- B2 deficiency may result in oxidative stress, mitochondrial dysfunction, low uric acid, low B3 or B6, high homocysteine, anemia or oral & throat inflammation.
- Food sources include milk, cheese, eggs, whole grains, beef, chicken, wheat germ, fish, broccoli, asparagus, spinach, mushrooms and almonds.

Folic Acid - B9



- Folic acid plays a key role in coenzymes involved in DNA and SAMe synthesis, methylation, nucleic acids & amino acid metabolism and RBC production.
- Low folate may result from alcoholism, high-dose NSAIDs, diabetic meds, H2 blockers, some diuretics and anti-convulsants, SSRIs, methotrexate, trimethoprim, pyrimethamine, triamterene, sulfasalazine or cholestyramine.
- Folate deficiency can result in anemia, fatigue, low methionine, increased homocysteine, impaired immunity, heart disease, birth defects and CA risk.
- Food sources include fortified grains, green vegetables, beans & legumes.

Niacin - B3



- B3 is used to form NAD and NADP, involved in energy production from food, fatty acid & cholesterol synthesis, cell signaling, DNA repair & cell differentiation.
- Low B3 may result from deficiencies of tryptophan (B3 precursor), B6, B2 or Fe (cofactors in B3 production), or from long-term isoniazid or oral contraceptive use.
- B3 deficiency may result in pellagra (dermatitis, diarrhea, dementia), neurologic symptoms (e.g., depression, memory loss), bright red tongue or fatigue.
- Food sources include poultry, beef, organ meats, fish, whole grains, peanuts, seeds, lentils, brewer's yeast and lima beans.

Cobalamin - B12

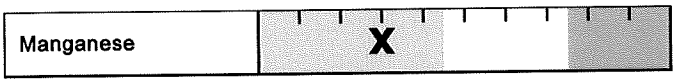
- B12 plays important roles in energy production from fats & proteins, methylation, synthesis of hemoglobin & RBCs, and maintenance of nerve cells, DNA & RNA.
- Low B12 may result from alcoholism, malabsorption, hypochlorhydria (e.g., from atrophic gastritis, H. pylori infection, pernicious anemia, H2 blockers, PPIs), vegan diets, diabetic meds, cholestyramine, chloramphenicol, neomycin or colchicine.
- B12 deficiency can lead to anemia, fatigue, neurologic symptoms (e.g., paresthesias, memory loss, depression, dementia), methylation defects or chromosome breaks.
- Food sources include shellfish, red meat poultry, fish, eggs, milk and cheese.

Amino Acids, 24hr Urine Interpretation At-A-Glance

Nutritional Needs

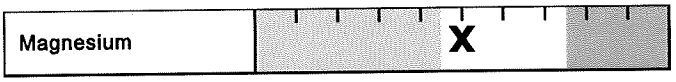
Minerals

Manganese



- Manganese plays an important role in antioxidant function, gluconeogenesis, the urea cycle, cartilage & bone formation, energy production and digestion.
- Impaired absorption of Mn may occur with excess intake of Fe, Ca, Cu, folic acid, or phosphorous compounds, or use of long-term TPN, Mg-containing antacids or laxatives.
- Deficiency may result in impaired bone/connective tissue growth, glucose & lipid dysregulation, infertility, oxidative stress, inflammation or hyperammonemia.
- Food sources include whole grains, legumes, dried fruits, nuts, dark green leafy vegetables, liver, kidney and tea.

Magnesium

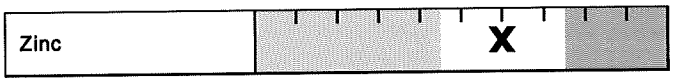


- Magnesium is involved in >300 metabolic reactions. Key areas include energy production, bone & ATP formation, muscle & nerve conduction and cell signaling.
- Deficiency may occur with malabsorption, alcoholism, hyperparathyroidism, renal disorders (wasting), diabetes, diuretics, digoxin or high doses of zinc.
- Low Mg may result in muscle weakness/spasm, constipation, depression, hypertension, arrhythmias, hypocalcemia, hypokalemia or personality changes.
- Food sources include dark leafy greens, oatmeal, buckwheat, unpolished grains, chocolate, milk, nuts & seeds, lima beans and molasses.

Molybdenum

- Molybdenum is a cofactor for enzymes that convert sulfites to sulfate, and nucleotides to uric acid, and that help metabolize aldehydes & other toxins.
- Low Mo levels may result from long-term TPN that does not include Mo.
- Mo deficiency may result in increased sulfite, decreased plasma uric acid (and antioxidant function), deficient sulfate, impaired sulfation (detoxification), neurologic disorders or brain damage (if severe deficiency).
- Food sources include buckwheat, beans, grains, nuts, beans, lentils, meats and vegetables (although Mo content of plants depends on soil content).

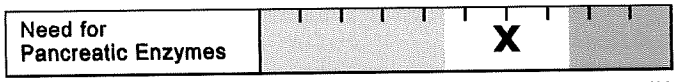
Zinc



- Zinc plays a vital role in immunity, protein metabolism, heme synthesis, growth & development, reproduction, digestion and antioxidant function.
- Low levels may occur with malabsorption, alcoholism, chronic diarrhea, diabetes, excess Cu or Fe, diuretics, ACE inhibitors, H2 blockers or digoxin.
- Deficiency can result in hair loss and skin rashes, also impairments in growth & healing, immunity, sexual function, taste & smell and digestion.
- Food sources include oysters, organ meats, soybean, wheat germ, seeds, nuts, red meat, chicken, herring, milk, yeast, leafy and root vegetables.

Digestive Support

Need for Pancreatic Enzymes



- Pancreatic enzymes are secreted by the exocrine glands of the pancreas and include protease/peptidase, lipase and amylase.
- Pancreatic exocrine insufficiency may be primary or secondary in nature. Any indication of insufficiency warrants further evaluation for underlying cause (i.e., celiac disease, small intestine villous atrophy, small bowel bacterial overgrowth).
- A high functional need for digestive enzymes suggests that there is an impairment related to digestive capacity.
- Determining the strength of the pancreatic enzyme support depends on the degree of functional impairment. Supplement potency is based on the lipase units present in both prescriptive and non-prescriptive agents.

Exhibit X

The Truth About Progesterone and Breast Cancer

By Elyn Jacobs

There's a lot of confusion out there when it comes to the topic of progesterone and breast cancer. About 70% of breast cancers are ER+ (estrogen receptor-positive), and most of these breast cancers (about 87%) are also PR+ (progesterone receptor-positive).

Hormone receptor status has long been a main factor in considering breast cancer treatment. Many women worry when they hear that their breast cancer is both ER+ and PR+, believing this to be "double trouble." It is time to set the record straight.

Actually, doctors have known for a long time that **women with high levels of both estrogen and progesterone receptors (high ER+ and PR+ status) often have the best chance of surviving**. Despite this, it appears that some oncologists may not really understand or explain one very key point...

While estrogen can fuel a tumor's growth, progesterone puts the brakes on that growth. Hence, they leave patients in a sea of fear and confusion unnecessarily.

What are Estrogen and Progesterone Receptors, and What Do They Do?

Estrogen and progesterone receptors are proteins found within many of the cells of our bodies, including cells in the breasts. Both receptors are directly involved in switching genes on and off – some 470 different genes. When estrogen and progesterone are present, these hormones stick to their respective receptor. They can then attach to specific regions of our DNA and turn genes on or off, changing the cell's behavior.

Hormone receptor-positive breast cancers have many hormone receptors. When breast cancer develops , the tumor cells become overly sensitive to estrogen. When estrogen activates the estrogen receptor, it turns on a panel of genes that tell the cells to keep dividing, driving tumor growth. However, when breast cancer cells have working progesterone receptors, and there is sufficient progesterone available, **progesterone will slow down estrogen fueled growth and division of these cells.**

The late John Lee, MD, author of *What Your Doctor May Not Tell You About Breast Cancer,* was on to this years ago. He maintained that when activated by progesterone, the progesterone receptors attach themselves to the estrogen receptors.

Once this happens, the estrogen receptors stop turning on genes that promote the growth of the cancer cells. Instead, they turn on genes that promote the death of cancer cells (known as apoptosis) and the growth of healthy, normal cells. Yet few seem to have been paying attention to his advice. Therefore, many doctors continue to villainize progesterone and progesterone status.

New Study Highlights Benefits of Progesterone

Hopefully a study published in the Dec 2016 edition of the journal *Nature,* led by Cambridge-based Cancer Research U.K. researcher Dr. Jason Carroll of the University of Adelaide in Australia, brings more credence and awareness to the benefits of progesterone and progesterone receptor-status.

The presence of both ER and PR status has typically been considered an indication of how good a woman's chances of surviving were. The belief being these cancers were more "treatable" than hormone receptor-negative cancers.

But in Dr. Carroll's study, the scientists confirmed the "why." They found that progesterone – via the progesterone receptor – is somehow affecting how the estrogen receptor works. Interestingly, they found that the progesterone receptor, in effect, "reprograms" the estrogen receptor, changing the genes that it influences.

But, the most important part was the overall effect this has on the cancer cells themselves. **Progesterone seemed to cause the cells to stop growing as quickly.** Dr. Carroll's findings further explain *why* the receptor itself is the direct reason why women who have both ER+ and PR+ have a better outlook than those with just ER+ or receptor-negative cancers.

The Role of HER2 and Progesterone in Breast Cancer

Two other recent studies suggest that metastatic dissemination of tumor cells prior to the detection of a primary tumor is regulated via HER2 expression and progesterone signaling. By implicating progesterone in the development of early metastasis, one could infer that progesterone is *bad.*

However, it is this author's opinion that **it is important to understand the difference between progesterone that is made by the human body and synthetic progesterone,** which is unnatural to the body.

Progesterone is a key physiologic hormone of women. But while natural progesterone has an anticancer effect, synthetic progesterone (found in birth control pills and hormone replacement supplements) does not. For example, research shows that the synthetic version progestin (medroxyprogesterone) is not only linked to breast cancer, but that those cancers tend to be "more aggressive and deadlier."

Furthermore, researchers have known for some time that synthetic progesterone does not stimulate activation of the tumor suppressor gene p53 when it attaches to progesterone receptors.

P53 is a repair gene, which protects cells from becoming cancerous. It is the primary gene that protects women from breast cancer. In order for progesterone to facilitate the production of p53, it must attach itself to progesterone

Exhibit X

P53 is a repair gene, which protects cells from becoming cancerous. It is the primary gene that protects women from breast cancer. In order for progesterone to facilitate the production of p53, it must attach itself to progesterone receptors.

If synthetic progesterone (again, which *does not* stimulate the production of p53) is present on the receptors, **natural progesterone will not be able to occupy the receptors.**

Clearly, it would be a good idea to down-regulate HER2, maintain healthy progesterone levels, and avoid synthetic progesterone. (While Herceptin is the drug of choice for HER2, daily consumption of 25 grams of flaxseed has been shown to decrease HER2 expression by 71%, which appears to outperform the drug, sans the damaging effects of drugs.).

Doing so could not only increase the chances of recovering from breast cancer, but could also help avoid getting breast cancer in the first place.

You are now empowered with the truth about progesterone and breast cancer. Please share this vital information with friends and family who could benefit.

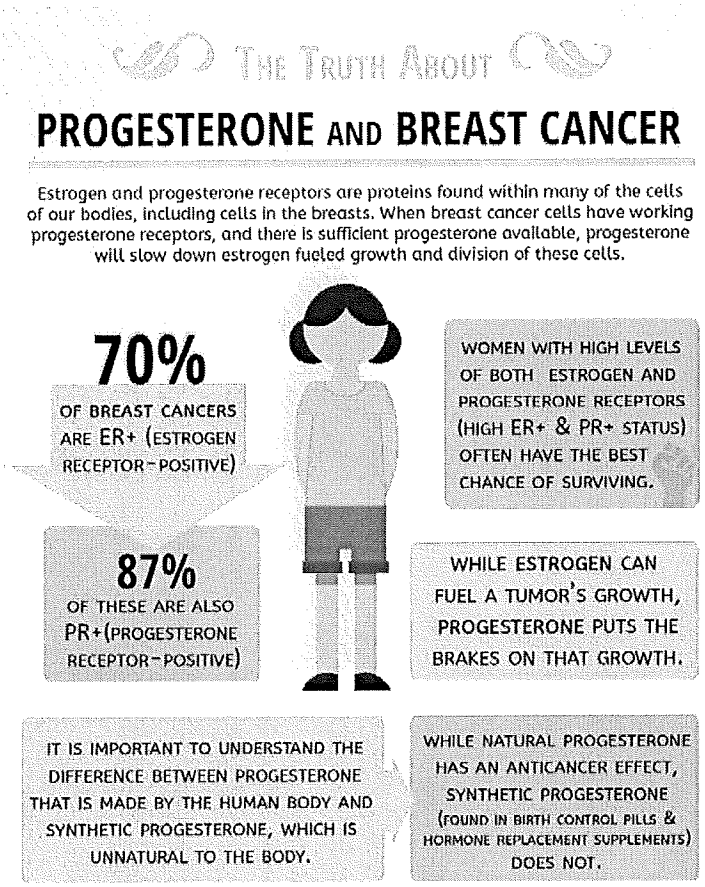


Exhibit Y
Why Low Progesterone May be Bad for You

Hormone balance is important. Any woman can tell you that she notices when her hormones are off. Imagine many of the diagnoses that women come to the doctor with that are related to an imbalance in hormones. Menopausal symptoms, hot flashes, night sweats, PMS, postpartum depression, and infertility; just to name a few. But, there are many more conditions related to hormone balance; some obvious, and some not. I think we are experiencing an epidemic of female conditions that are specifically related to low progesterone or similarly, elevated estrogen.

What does Progesterone Do?

Progesterone is such an important hormone in mood and sleep and overall sense of wellbeing. Progesterone helps you maintain your level-headedness. In the body, progesterone is responsible for preparing the lining of the endometrium to get ready for implanting a fertilized egg, therefore progesterone is extremely important in the success of conception. Each month when this doesn't happen, the lining is shed. Progesterone also helps balance blood sugar levels, boosts and regulates thyroid function, helps the break down of fat to be used as energy, is anti-inflammatory in nature as it helps to reduce swelling and inflammation.

Progesterone Symptoms

A few of the symptoms of low progesterone are sugar cravings, ovarian cysts, irregular periods, recurrent early miscarriage, blood clots, cold hands and feet, decreased sex drive, depression, anxiety, mood swings, acne, insomnia, constant allergies, fatigue, endometriosis, short luteal phase, infertility, and slow metabolism

Estrogen Dominance

Many conditions are termed "low progesterone" but may in fact not be related to low progesterone. Many times, when someone has what we call estrogen dominance, they have elevated estrogen relative to how much progesterone they have. So this means that someone can have low progesterone symptoms with normal progesterone levels. The reason this happens is because when your estrogen is high, your body senses that your progesterone is low, even if it is not. Therefore your body will trigger symptoms of low progesterone because of the excess of estrogen.

Estrogen dominance, or excess estrogen, is happening more commonly due to our increase in exposure to estrogens. We get exposed to them environmentally as well as in some water supplies due to the abundance of estrogens being taken orally by women. If a woman is using a form of topical estrogen in the home, tests have found elevated estrogen in family members as estrogen is easily transferred from the hands of one to another. Environmentally, we have accumulated excess estrogen-like compounds throughout our years of using plastics. These chemicals are called xeno-biotics and they fit similarly into estrogen receptors, making the body sense extra estrogen and sometimes even driving some metabolic processes within the body.

In addition, we are becoming larger as a culture and with additional fat cells, we tend to accumulate more estrogen. In an overweight individual, this excess estrogen can lead to low progesterone or low progesterone symptoms.

True Dangers of Low Progesterone

My concern for the low progesterone/ high estrogen problem is that we are seeing the imbalance leads to some dangerous conditions such as breast cancer, ovarian cancer, prostate cancer, and possibly uterine cancer. Excess estrogen, without the balance of progesterone, can be dangerous if left untreated.

I am not saying that everyone needs to take progesterone, but every woman should be aware of what her body is telling her. Early symptoms are the best way to determine E:P balance. Estrogen to progesterone imbalance can be discovered from a very young age and it can be treated and prevented early. So, for instance, if I see a 14 year old girl having significant cramping, heavy bleeding, and PMS, my first line of treatment is to balance the E:P ratio so that she starts having a more comfortable cycle. This is what is important to her. But what is important to me, but maybe I haven't shared this with her, is that in keeping her hormones balanced when she is younger, she is going to have a better transition into fertility, have healthier balanced pregnancies, can go into menopause more smoothly, and have a better chance at preventing breast or ovarian cancer. This is a lot to think about for a 14 year old, but when balancing a girl's hormones, I can't but think of her future and feel grateful that I can be a part in her prevention.

Stress Causes Low Progesterone

Another reason progesterone may be low is when a person is under stress. You may recall someone under a great deal of stress who may have missed one or a few cycles. This occurs because the precursor hormone that is used to make progesterone and estrogen also has to make the stress hormone. When someone is under stress for any reason, stress always wins. We were designed that way evolutionary because the types of stressors we came across were significant such as the common example of running from the tiger. We have not evolved past this time as of yet, therefore living under constant stress as many people do, has certainly taken a toll on physiology and how all of the organs of the body function together. The stress glands, the adrenals, always win and usually what organs suffer are the ovaries and the thyroid.

Taking progesterone is not always the best solution, depending on many factors. Age, whether one wants to get pregnant in the near future, family history of progesterone sensitive breast cancers, and other precautions must be taken before just starting progesterone. Herbs and dietary habits can help with estrogen dominance and reducing stress can help with stress induced low progesterone. Decreasing inflammation through diet and improving blood sugar can help ward off estrogen dominance. Here are some dietary suggestions below.

Dietary Suggestions for Reducing Estrogen Dominance:

Eliminate all sugar

Eliminate all juice

Avoid alcohol

Consume only 1-2 fruits per day

Do no consume any dried fruit

Drink ½ your weight in ounces daily of filtered or bottled water

Eliminate all pastries, cookies, breads, donuts, and cakes

Only consume organic animal products and reduce them in your diet

Eat walnuts for reducing inflammation and increasing omega 3 fatty acids

Eat ground flax seeds for increasing sex hormone binding globulin

Eat hemp seeds for reducing inflammation and increasing omega 3 fatty acids

Eat vegetables, especially cruciferous vegetables such as cabbage, broccoli, Brussels sprouts and cauliflower

Rid your diet of all processed foods. No boxes.

Stress Relief Suggestions:

Try meditating daily, even if it is 5 minutes per day. Sit in a comfortable position and concentrate and bringing air in through the nose and out through the mouth. Practice mindfulness and keep yourself in a place of non-judgment

Drink chamomile tea after dinner

Take 400 mg of magnesium after dinner

Begin to visualize happy thoughts daily

Progesterone has Influences Throughout the Body

The sooner you start to work on hormone balance, the better you will feel. Because of progesterone's widespread functions throughout the body, working towards better hormone balance between estrogen and progesterone is also important to any other symptoms you may be experiencing. I once treated a woman who came in with endometriosis and a seizure disorder. Since I didn't know how to start approaching her seizures (this was early on in practice), I told her we were going to work on her endometriosis first. After we treated her endometriosis, her seizures spontaneously stopped.

Exhibit Z

Low Progesterone: Complications, Causes, and More

Medically Reviewed by Debra Rose Wilson, PhD, MSN, RN, IBCLC, AHN-BC, CHT on August 25, 2016 — Written by Ana Gotter

Low progesterone Progesterone levels Treatment Outlook

What is progesterone?

Highlights

Low progesterone levels can make it difficult for women to get pregnant or carry a baby to full term.

Symptoms of low progesterone include low sex drive, migraines, hot flashes, and abnormal uterine bleeding.

Your progesterone levels can be determined through a simple blood test.

Progesterone is a female sex hormone produced mainly in theovaries following ovulation each month. It's a crucial part of the menstrual cycle and maintenance of pregnancy.

Progesterone helps to regulate your cycle, but its main job is to get your uterus ready for pregnancy. After you ovulate each month, progesterone helps thicken the lining of the uterus in preparation for a fertilized egg. If there is no fertilized egg, progesterone levels drop again and menstruation begins. If a fertilized egg does implant in the uterine wall, progesterone helps to maintain the uterine lining throughout pregnancy.

Progesterone is necessary for breast development and breast-feeding as well. It complements some of the effects of estrogen, another female hormone. It also plays an important role withtestosterone, as it is the precursor for adrenal hormones.

Men produce a small amount of progesterone to aid in the development of sperm.

LOW PROGESTERONE

Should I be concerned about low progesterone?

Progesterone is especially important in your childbearing years. If you don't have enough progesterone, you may have trouble getting or staying pregnant. Each month, after one of your ovaries releases an egg, your progesterone levels should rise. Progesterone helps the uterus thicken in anticipation of receiving a fertilized egg. If it's not thick enough, implantation doesn't occur.

Symptoms of low progesterone in women who aren't pregnant include:

- headaches or migraines
- mood changes, including anxiety or depression
- low sex drive
- hot flashes
- irregularity in your menstrual cycle

For women who aren't pregnant, low progesterone may cause abnormal uterine bleeding. Irregular or absent periods may indicate poorly functioning ovaries and low progesterone.

If you do get pregnant, you still need progesterone to maintain your uterus until your baby is born. If your progesterone levels are too low, your uterus may not be able to carry the baby to term.

During pregnancy, symptoms of low progesterone include spotting and abdominal pain. Other symptoms of low progesterone in women who are pregnant may include:

- constant breast tenderness
- unrelenting fatigue
- frequent low blood sugar
- vaginal dryness

Low progesterone may indicate toxemia or ectopic pregnancy. This can sometimes result in miscarriage or fetal death.

Without progesterone to complement it, estrogen may become the dominant hormone. This may lead to a variety of symptoms, including:

- weight gain
- decreased sex drive, mood swings, and depression
- PMS, irregular menstrual cycle, heavy bleeding
- breast tenderness, fibrocystic breasts
- fibroids, endometriosis
- gallbladder problems
- thyroid dysfunction

Learn about fibroids »

PROGESTERONE LEVELS

Understanding and testing levels

A progesterone test (PGSN) can help your doctor determine if your progesterone levels are too low. This is a simple blood test that doesn't require any preparation.

The test can offer clues as to why you're having trouble getting pregnant. It can also confirm whether or not you've ovulated. The PGSN test can be used to monitor hormone replacement therapy or

the health of a high-risk pregnancy.

Progesterone levels fluctuate throughout the menstrual cycle. They peak about seven days before your period, and can even vary in the course of a single day. Progesterone levels are usually higher than normal during pregnancy. They're even higher if you're expecting more than one baby.

Poorly functioning ovaries can result in poor progesterone production. During menopause, it's natural for estrogen and progesterone levels to fall.

Men, children, and postmenopausal women all have lower progesterone levels than women in their childbearing years.

What is considered a "normal" progesterone level depends on a person's age and gender. In women, additional factors include whether she's pregnant and where she is in her menstrual cycle.

TREATMENT

What can I do about low progesterone?

Having low progesterone may not have any symptoms for you, and you may not need to treat it. However, if you're trying to have a baby, hormone therapy to increase progesterone may help thicken your uterine lining. This may improve your chances of having a healthy pregnancy and carrying to term.

Menstrual irregularities and abnormal bleeding can also be improved through hormone therapy.

For severe symptoms of menopause, hormone therapy usually consists of a combination of estrogen and progesterone. Women who take estrogen without progesterone are at increased risk of developing endometrial cancer.

Treatment options include:

- creams and gels, which can be used topically or, in women, vaginally
- suppositories, which are commonly used when treating low progesterone that is causing fertility problems
- vaginal rings, which offer slower releases of hormones than oral medications
- oral medications, like Provera

Hormone therapy may help ease symptoms such as hot flashes,night sweats, and vaginal dryness. For some women, it improves mood and state of mind. It may also lower your risk ofosteoporosis and diabetes. Oral progesterone may provide a calming effect, making it easier to sleep.

Hormone therapy may increase the risk of stroke, blood clots, and gallbladder troubles. If you've had breast cancer or endometrial cancer, your doctor will probably advise against hormone therapy. Women with a history of liver disease, blood clots, or stroke shouldn't undergo hormone therapy.

Read more about hormone replacement therapy »

Natural remedies for raising low progesterone levels include:

- increasing your intake of vitamins B and C, which are necessary for maintaining progesterone levels
- eating more foods with zinc, like shellfish
- controlling stress levels, as cortisol is released instead of progesterone in high periods of stress and it reduces progesterone levels

Progesterone is still important in women who have hadhysterectomies. Women who have had hysterectomies and are not on progesterone replacement are more likely to die from heart disease and have increased risk for both brain and bone diseases.

Because women with hysterectomies are also more likely to have strokes or cancer while on conventional hormone therapies, undergoing bioidentical hormone therapy is essential. In bioidentical hormone therapy, the hormones you're receiving are biologically identical to your natural hormones. Women on this type of hormone therapy have no increased risk of disease.

OUTLOOK

Outlook

Low progesterone can cause a number of different problems for both men and women. However, there are several different types of treatments that can help resolve low progesterone, allowing you and your doctor to choose which solution is best for you. It may take a few weeks before you see results from hormone therapy.

Hormone therapy may be a long-term solution for some, particularly postmenopausal women. You can work with your doctor to re-evaluate your treatment plan each year.

Article resources

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Printed On Sep 27, 2007

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Urgency: Routine
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Provisional Diagnosis: hair breakage
Reason For Request:
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hair breakage, little body hair; more facial hair
? vitamin problems leading to above (or other considerations you migh t have)

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Last Action: PRINTED TO

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